VOLUME 50 NUMBER 2

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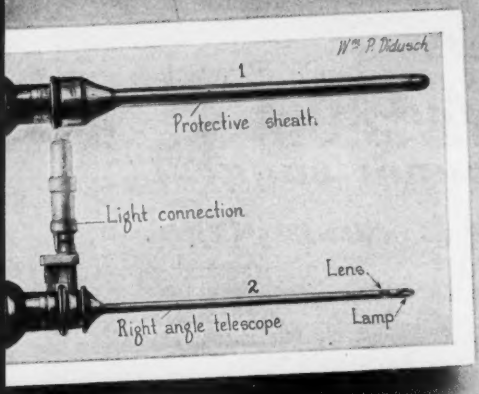

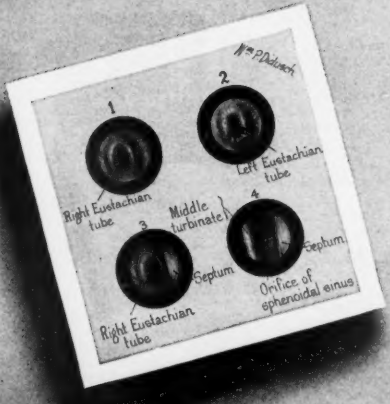

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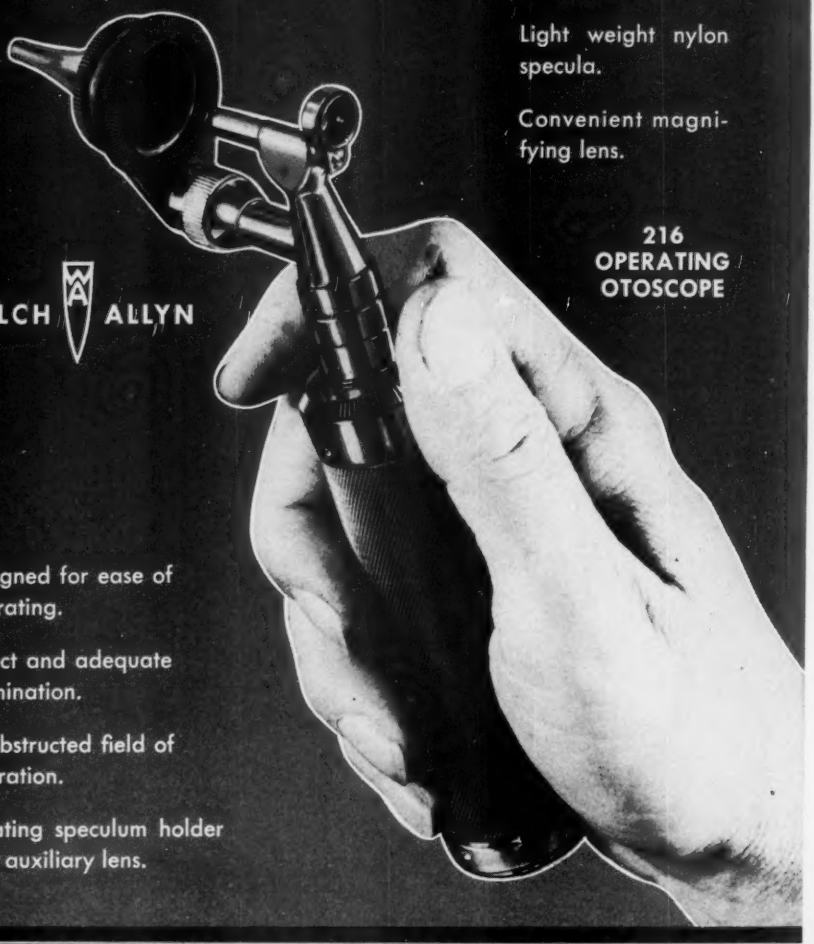

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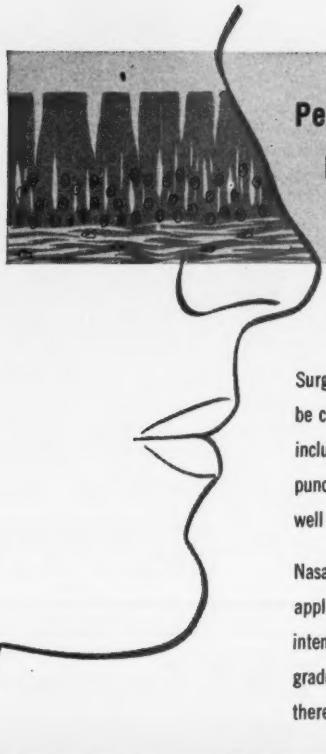

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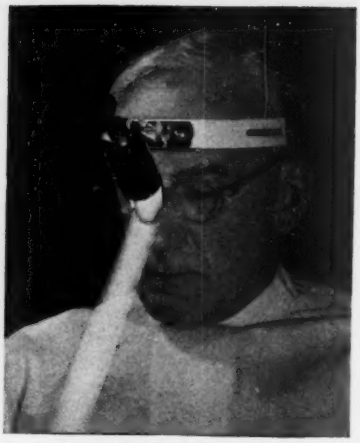

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OTOSCLEROSIS: THEORY OF ITS ORIGIN AND DEVELOPMENT

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AND
DOROTHY WOLFF, Ph.D.
NEW YORK

IN THE May 1945 issue of the ARCHIVES OF OTOLARYNGOLOGY we published a report of our histologic study of the incus and the head and neck of the malleus. These ossicles were removed during the performance of the fenestra nov-ovalis operation in 115 cases of ankylosis of the stapedial footplate caused by a proliferating otosclerotic lesion in the region of the oval window. Attention was called to the fact that a single case of otosclerosis involving the malleus and the incus, as proved in microscopic sections, had been reported and illustrated in the literature.[1] The object of our study was to determine how frequently otosclerosis involves the incus and the malleus in the presence of an otosclerotic lesion within the otic capsule.

We reported that in the series of ossicles examined, no typical circumscribed focus of otosclerosis was observed. No pathologic area clearly demarcated from adjacent normal bone was found. No area such as has been seen in the region of the oval window or elsewhere in the otic capsule could be demonstrated in the two temporal bones from the 2 patients who died from irrelevant causes after undergoing the fenestration operation for clinical otosclerosis. We did, however, observe numerous pathologic changes in the joints, in the marrow spaces and in the bone of these ossicles and particularly in the blood vessels thereof. In fact, completely normal ossicles were found in only 5 of the 115 cases examined.

A histologic study of a second series of ossicles removed from 100 patients whose labyrinths were being fenestrated for clinical otosclerosis is herewith presented. In this study the incus, the head and neck of the malleus and occasionally the head and crura of the stapes were included. As in the preceding series, no typical circumscribed focus of otosclerosis was found. No clearly demarcated pathologic area comparable to that occurring in the otic capsule of the otosclerotic

From the Lempert Institute of Otology and the Lempert Research Foundation, Inc.

1. Covell, W. P.: The Ossicles in Otosclerosis, Acta oto-laryng. **28**:263, 1940.

patient could be demonstrated. As in the preceding series, numerous pathologic changes were observed, with completely normal ossicles being found in only 4 cases. Again pathologic changes were found in the joints, the bony tissue and the marrow spaces of the ossicles and particularly in the blood vessels.

COMMENT

Since the pathologic changes observed in the ossicles removed in vivo from patients suffering from clinical otosclerosis were identical with those observed in the ossicles of postmortem material wherein the lesion of otosclerosis was observed in the otic capsule (see figs. 29 to 32), we felt that the lesions occurring in the ossicles, though not circumscribed, were part of the otosclerotic process observed in the otic capsule. We believe that the reason the lesions in the ossicles are not circumscribed by healthy bone, whereas in the otic capsule they are, is that the number of blood vessels normally feeding the ossicles is limited as compared with the much richer blood supply normally nourishing the otic capsule. The ossicles receive their blood supply from one or at most two sources,[2] and the otic capsule from six sources [3] (perhaps more).

One of the outstanding observations made in the histologic study of these two series of ossicles was that the blood vessels constantly show pathologic involvement and that this is similar to the pathologic involvement of the blood vessels of otosclerotic foci observed within the otic capsule. Since these ossicles were always removed in vivo and did not undergo postmortem changes, we felt that an unusual, heretofore unobtainable, opportunity for careful study of the blood vessels and their contents was presented in this second series of 100 ossicles. Furthermore, we felt that by carefully observing the pathologic changes in the blood vessels and correlating them with the pathologic changes in the bony structure of the ossicles we could perhaps determine whether the vascular changes were not most likely the forerunners of, and responsible for, the pathologic changes in the bony structure known as otosclerosis.

VASCULAR ANATOMY OF THE OSSICLES HISTOLOGICALLY TRACED

In a study of the vascular supply of the normal ossicles attention must be given to the fact that we are dealing with the smallest bones of the body. The caliber of the vessels concerned is therefore a matter of relativity. Vessels ramifying through these bones and

2. Nabeya, D.: The Blood-Vessels of the Middle Ear, in Relation to the Development of the Small Ear-Bones and Their Muscles, Folia Anat. japon. **1**:243, 1922-1923.

3. Bast, T. H.: The Blood Supply of the Otic Capsule, Anat. Rec. **48**:141, 1931.

establishing haversian systems are of necessity only of the caliber of the arterioles or even of the arteriolar capillaries found elsewhere in the body. Since there is but little need for muscular walls, muscle cells are but seldom seen in these minute vessels. Most of the vessels seem to depend solely on an endothelial wall. Interestingly enough the long axes of the nuclei of the endothelial cells appear to lie not only longitudinally with the course of the vessel but also circularly. This anatomic arrangement suggests a functional response, if not to neural stimulation, then to chemical stimulation. The possibility that pericytes are present as an aid to contraction must also be considered.

Some of the blood vessels ramifying through these bones take origin in their own local marrow spaces. These, as previously described, occur in the body of the incus and in the head of the malleus. The descending vessel of the descending process of the incus has been traced in many ossicles to this source. But these marrow spaces must of course maintain some connection with the vascular supply of the rest of the body. Accordingly, each ossicle has a nutrient artery coming as a branch of the middle meningeal artery.

The nutrient vessel of the incus enters the ossicle on the anterior surface just below the articular surface. The nutrient vessel of the malleus enters the neck of that ossicle.

Additional vessels may occasionally be observed passing from the mucosa to the bone or vice versa. It is difficult to know in which direction the vascular flow may be in vivo in these minute channels since there is no observable change in caliber. One such vessel was frequently observed by us to cross from the mucosa to the bone or vice versa on the superior surface of the malleus. Another was noted at the lower extremity of the descending process of the incus. In several instances the descending vessel of the descending process of the incus was observed to travel completely through the bone and emerge in the connective tissue of the submucosa at the lower extremity. Whether this descending arteriolar capillary ever entered its accompanying vein and allowed its contents thus to return to the marrow space is uncertain.

As stated, the study of the two series of ossicles routinely showed involvement of the blood vessels. An especial effort was therefore made to demonstrate their condition in the second series of ossicles. To this end the vessels in the marrow spaces were examined, the vessels in the descending process of the incus were studied, and those in the short process of the incus were studied, as were also those in the head and neck of the malleus. Those in the neck and the crura of the stapes were also examined when these were present. The vessels in the mucosa covering the ossicles were observed when that membrane was intact.

Within a haversian canal the venule accompanying the arteriole or arteriolar capillary may be three to five times the caliber of the arteriole. This is well illustrated in the apparently normal vessels of the descending process of the incus seen in figure 1.

Fig. 1.—Normal arteriole and venule in the descending process of the incus. The venule may be three to five times the caliber of the arteriole. Numerous polymorphonuclear cells are present in the venule. W. F., and adult, specimen 11.

THEORY OF ORIGIN AND PROGRESSIVE DEVELOPMENT OF OTOSCLEROSIS

After carefully studying and analyzing the pathologic changes in the second series of 100 ossicles, we shall present those changes which appear to us to be various stages of the otosclerotic processes in what

we believe to be the order in which the pathogenesis of otosclerosis most likely occurs:

We shall first consider the contents of the blood vessels, then the walls of these vessels within the ossicles, and finally the gradual and insidious changes in the bone itself, which appear to arise from the pathologic conditions of the blood vessels.

Fig. 2.—The arrow indicates the clear globules present within a vessel. In the haversian canal in the upper left field, two globules have emerged from the vessel and are becoming distorted in shape as from the absorption of a gas.

Changes in Vascular Contents.—Congestion and stasis [4] have long been observed within the vessels in otosclerosis. In the present study

4. Wittmaack, K.: Die Ursache der Otosklerose: Ein Vorschlag zur ursächlichen Behandlung, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:150, 1931.

we desired to observe, if possible, the contents of the congested vessels and to study the preliminary stages of the production of the congestion and stasis.

1. Clear globules. A quite unanticipated finding was that of clear globules commonly occurring within the lumen of the vessels. In 23 per cent of the cases these were observed in areas other than the marrow spaces. Vessels in the marrow spaces were not included in the count, because it was felt that in such vessels the globules might merely represent fat that was being transferred to or from the marrow

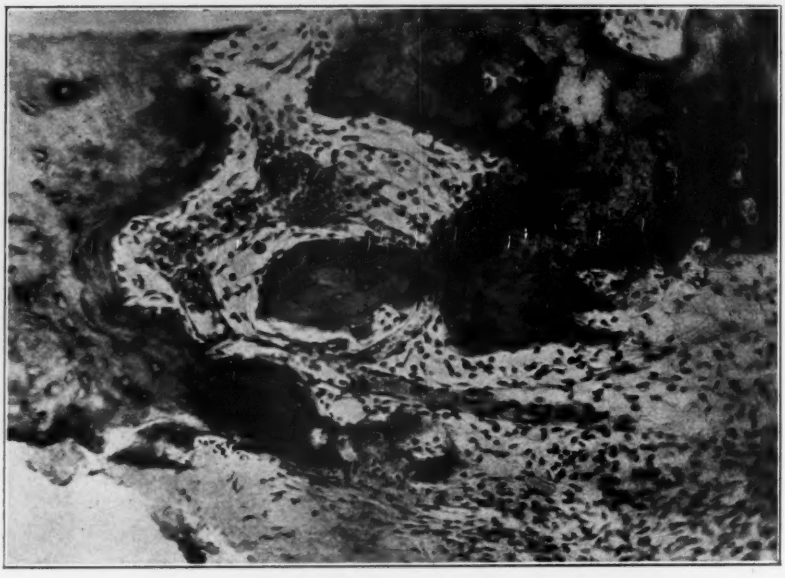

Fig. 3.—An island of bone is being formed in the process of pathologic decalcification. Note the evidence of congestion and stasis. Clear globules may be seen within the vessel and emerging through the wall. W. M., aged 28; specimen 1585.

space. The globules sometimes were so abundant as to fill more or less completely portions of the vessels; sometimes they were alined along the vessel walls. Figure 2 shows these globules within vessels in the upper center field. In figure 3 the globules are seen in the act of emerging through the vessel wall and escaping into the surrounding tissue. Once having escaped, they do not maintain their globular shape but become distorted. Their appearance is that of a balloon absorbing gas. Figures 13 and 18 illustrate, in their center fields, the distending type of globule.

Such globules were photographed and reproduced without comment in our previous paper concerning ossicles, in figures 16 C and 18. Examination of microscopic sections of ossicles from patients with clinically diagnosed otosclerosis of the petrosa, as well as of otosclerotic centers from patients without clinically diagnosed oto-

Fig. 4.—Sludged blood in a tortuous vessel in the neck of the malleus. W. M., aged 24; no. 2272.

sclerosis but with the typical microscopic lesion, revealed that clear globules are numerous within the vessels in the involved area. A question arises as to the significance of these globules. Do they represent the beginning of edema in the tissue or are they a part of the process of saponification and decalcification?

2. Accumulating fragmented red blood cells. These were observed within the lumens of vessels in 27 per cent of the cases. They occurred in both arterioles and venules. They were often seen in areas of excessive constriction and hypertrophy of endothelial cells. But since they were also seen in widely patent veins, it seems likely that a chemi-

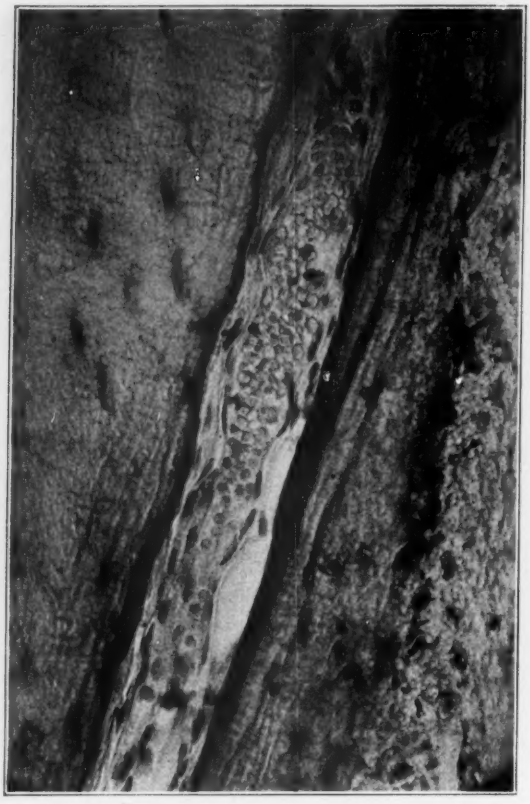

Fig. 5.—Early periodic constrictions of a vessel in the descending process of the incus. Note the two clear spaces to the right of the vessel. W. F., aged 52; no. 543.

cal condition caused their disintegration. This might have been lack of oxygen and accumulation of carbon dioxide. They were often found in the neighborhood of numerous polymorphonuclear cells.

3. Sludge. Clusters of misshapen red blood cells were observed agglutinated to each other, each aggregation separated from the succeed-

ing one by a clear space. Occasionally the clusters were agglutinated to the vessel wall. More often they were free in the lumen. The appearance was strikingly like that of the sludged blood seen in movies of living human and animal tissues. This seemed to be a "still" picture of the same process. It was observed in 21 per cent of the cases. Only 3 of

Fig. 6.—Early varicosities of the vein of the descending process of the incus. W. F., aged 20; no. 1269.

these cases fell into the group in which the globules were seen within the lumen. Sludged blood is illustrated in figure 4.

4. Accumulating polymorphonuclear leukocytes. These cells were predominantly the mature form of polymorphonuclear neutrophils, although band forms occurred They were observed in 40 per cent of

the ossicles. Round cells were also observed, but not nearly so often. Considering the chronicity of the disease the presence of polymorphonuclear cells is somewhat surprising. It is possible that they were called forth as a result of the inflammation arising from the localized operative procedure. In figure 1 they are seen in an otherwise normal vessel in the descending process of the incus.

Changes in the Vessel Walls.—1. Constriction. Some vessels appeared slightly constricted at regular intervals. Where no actual pathologic change of either the wall or the surrounding tissue was

Fig. 7.—Note the changing caliber of the arteriole as it crosses the marrow cavity. In the upper right corner of the field it is expanded. In the center field it is constricted, allowing the passage of only a row of red blood corpuscles. In the lower left field no lumen is apparent. W. F., aged 52; no. 1178.

noted, the constriction may have represented the normal rhythmic action caught by the surgeon at the time of removal of the ossicles.

(*a*) Constriction of venule: Figure 5 illustrates periodic constriction of the walls of a venule of the descending process of the incus. Evidence of incipient pathologic change may be observed in the two elongated clear areas to the right in the vessel. These represent either the first evidence of edema or gas absorbed by certain clear globules as described in foregoing paragraphs. In fact, they may prove to be the microscopic picture of both of these conditions.

More extensive constriction of a venule was found to lead to dilation of the vessel above and below the constricted area. Such a condition may be observed in figure 6, where early varicosities appear in the vein of the descending process of the incus of a 20 year old girl. The con-

Fig. 8.—A descending vessel of the descending process of the incus, reduced to a mere strand by complete constriction. Note the red blood cells in the patent portion in the upper right field. Three Howship's lacunas are seen in the bone bordering the vascular channel in the lower center field. W. M., aged 22; no. 1333.

comitant pathologic stasis, long recognized as a part of the microscopic otosclerotic picture, is here apparent.

(*b*) Constriction of arteriole: In figure 7 the constriction of an arteriole as it passes through a marrow space is clearly illustrated.

Complete constriction of an arteriole is again illustrated in figure 8, here occurring in the descending vessel of the descending process of the incus.

In regard to the matter of constriction of a vessel one must take into account the length of time the constriction lasts. In our study it

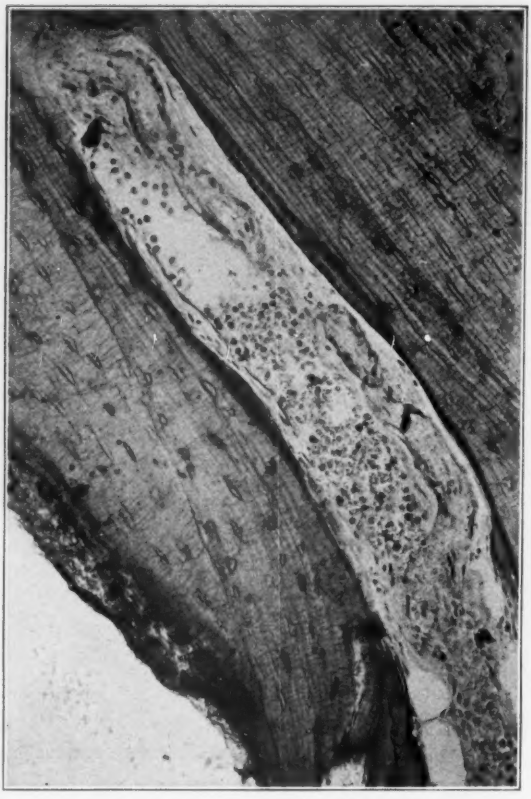

Fig. 9.—Periodic constriction and partial obliteration of the descending arteriole of the descending process of the incus. W. F., aged 45; no. 1308.

is impossible to observe the time factor, but the consequences of prolonged constriction may be seen. (Compare figure 24 with figure 8 to realize the nature of the pathologic changes occurring throughout this ossicle as a result of the pathologic changes occurring in the individual vessels.)

Periodic constriction of long standing is well shown in figure 9. Here in two of the constricted areas obliteration of the vessel is occurring. Of course, one must study serial sections to be sure of such an interpretation of this condition.

2. Tortuosity. In the preceding communication on ossicles we mentioned tortuosity of vessels as being most conspicuous among the

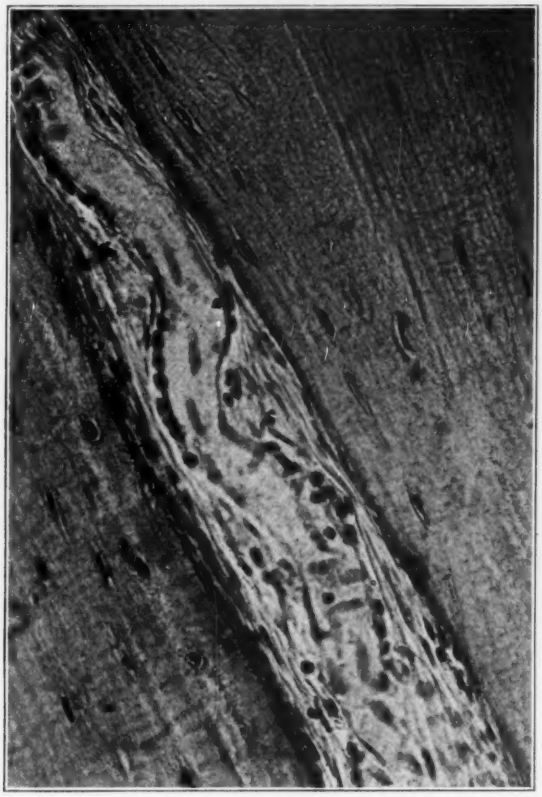

Fig. 10.—The descending vessel of the descending process of the incus, showing intermittent hypertrophy of endothelial cells. Note the pull of the vessel toward the hypertrophied cells, thus initiating tortuosity. W. F., aged 34; no. 549.

early vascular changes. In the present study we have attempted to find the incipient stages in the changes of the walls of the vessels leading to the production of tortuosity. In figure 10 we illustrate a very early stage in the development of tortuosity.

Hypertrophy of endothelial cells is obviously causing this condition. The hypertrophy occurs intermittently on one wall and in the intervening region on the opposite wall. The result is that a torque or pull of the vessel wall occurs toward the side of the hypertrophied cells, and tortuosity is produced. The possibility that pericytes are present on these nonmuscular walls has been mentioned. In figure 10 two apparently multinucleate structures appear on the right wall of the vessel. We are inclined to believe these are contractile processes of a pericyte coming into view and not multinucleate giant cells or osteo-

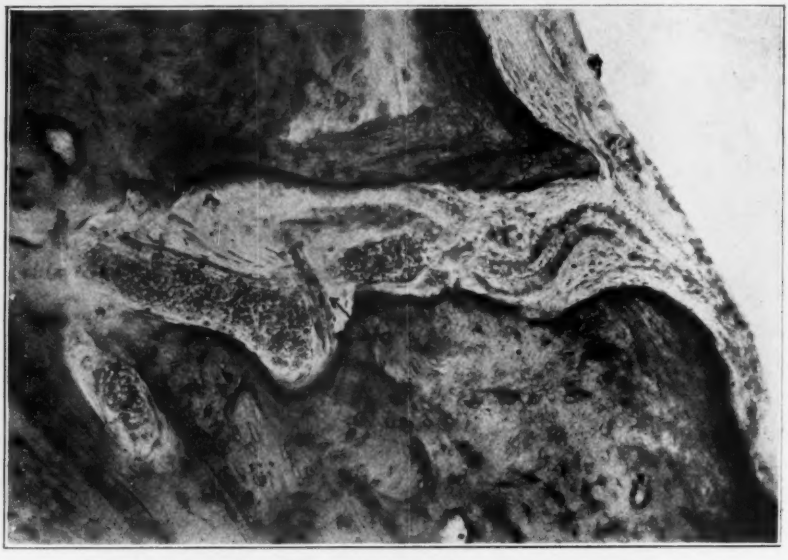

Fig. 11.—Hypertrophy of the endothelial cells passing into the head of the malleus. Note the tortuosity of the vessel. Fragmentation of red blood cells is present. W. F., aged 52; no. 1178.

clasts—which, incidentally, have been conspicuously absent in the sum total of these 100 ossicles.

In figure 11 one sees hypertrophy of endothelial cells producing marked tortuosity of the vessel and obliteration of the lumen, this last process resulting in obliteration of a part of the vessel itself.

The ultimate extent to which tortuosity of vessels can progress is illustrated in figure 12, a vessel in the descending process of the incus. Tortuosity of venous walls develops mostly from varicosities such as were described and illustrated in figure 6.

3. Periarteritis. This condition was described in our previous paper as occurring in 24 per cent of the cases. The condition might also be described as perivascular fibrosis, since it may arise around the venules as well as around the arterioles. This type of pathologic change occurred in the marrow spaces in 32 per cent of the cases and in the descending vessel of the descending process of the incus in 27 per cent of the cases. Only in 9 of the cases was this kind of pathologic condition exhibited in

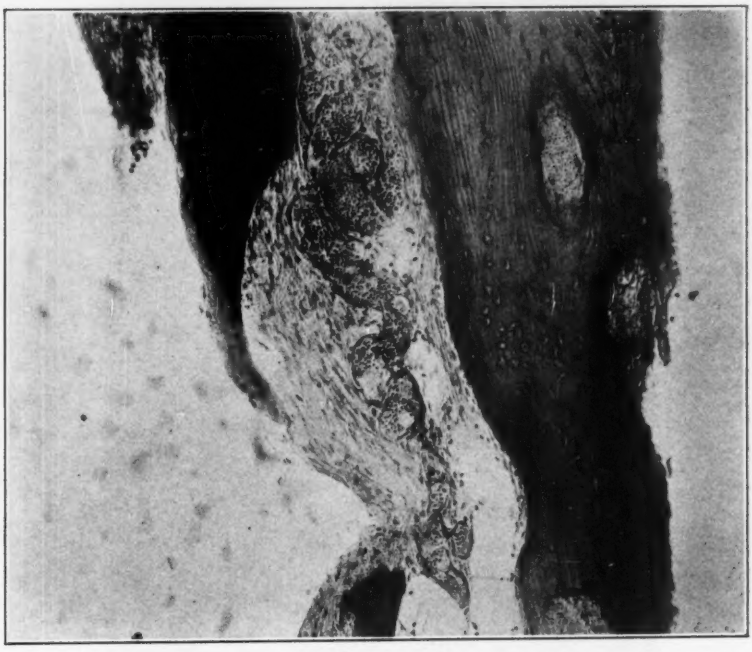

Fig. 12.—A tortuous vessel in the descending process of the incus. W. M., aged 63; no. 2096.

both of these regions. Figure 13 shows this lesion in the descending process of the incus.

4. Endarteritis. Obliterative endarteritis is well illustrated in figure 14. In three different areas in this field proliferation of the cells of the inner wall of these minute vessels may be observed. In the upper right the lumen of the vessel is only partially obliterated. In the middle and lower fields the lumen of the vessel is completely occluded. The ultimate consequences of this type of pathologic change are seen in figure 23.

Pathologic Changes of Bone.—1. Blue mantles. Blue mantles have long been described as a part of the microscopic picture of otosclerosis (fig. 15). The subject was discussed in our previous communi-

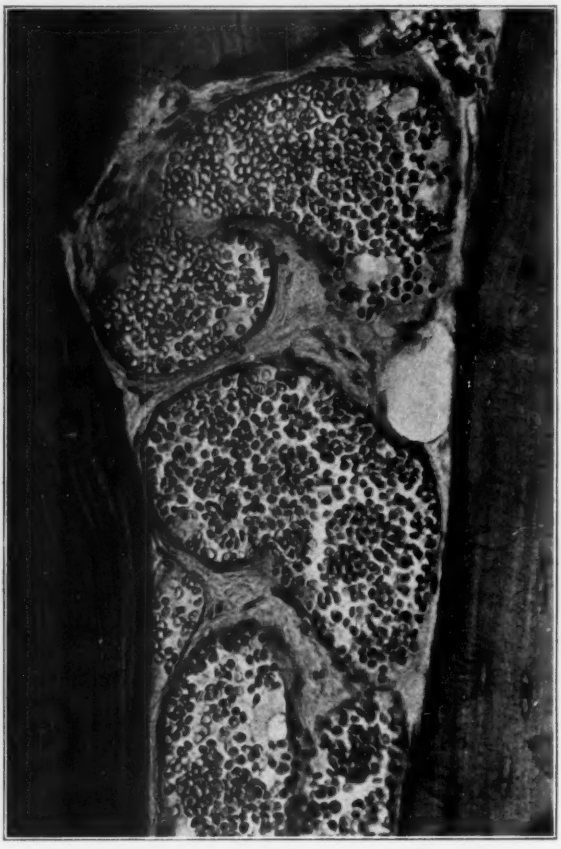

Fig. 13.—Perivascular fibrosis around the descending vessels of the descending process of the incus. Note the tortuosity, congestion, stasis and presence of clear globules. These are within the vessels, and one greatly dilated globule is in the surrounding tissue (center field). W. F., aged 54, no. 1233.

cation. The general microscopic appearance of bone may be altered by the contents of the blood stream. Blue mantles are the walls of haversian canals which take an intensely blue dye with the hematoxylin.

Blue mantles may occur in bone that is not otosclerotic, but they are always conspicuous in bone that is from patients with otosclerosis. They may, in a sense, be considered as chemical indicators of the type of calcium that has most recently been deposited along a particular blood

Fig. 14.—Detail showing obliterative arteritis. The vessel in the upper right corner shows partial obliteration due to hypertrophy of the endothelium. Periarterial fibrosis is also present. In the center and in the lower left corner complete obliteration of small vessels has occurred. Numerous lacunas devoid of bone cells give evidence of necrosis. W. F., aged 36; no. 12.

vessel. The spread of fluids emerging from the blood stream may be traced by the chemical reaction occurring in the bone along the canaliculi immediately adjacent to a haversian canal. In fact, there is a time in the

course of pathologic changes of bone when the row of lacunas lying immediately parallel to a haversian canal appear under the microscope like insects walking along a stick. At a later time the canaliculi on the other side of the lacunas will spread the devastating fluid to more distant lacunas. As this occurs, marked irregularity of staining is evident

Fig. 15.—Blue mantles along the vascular channels of the incus. They are more conspicuous in ossicles from otosclerotic patients. W. F., aged 28; no. 2087.

(fig. 16). The colors with hematoxylin and eosin staining may range in the same section from a grayish pink to a dark purplish blue in the bone itself. Areas of pale-staining bone may lie adjacent to areas showing excessive affinity for the dye or they may be adjacent to areas

giving a perfectly normal staining reaction. The more deeply staining bone may present a "seedy" or "peppery" appearance (fig. 17). What are the underlying causes of these varying hues? Selective affinity for dyes is known to be related to the chemical content of tissues. Vascular

Fig. 16.—Note the irregular staining reaction of the bone in this incus, particularly along the superior border. In the lighter areas many lacunas are devoid of bone cells, indicating necrosis. In the marrow space venous stasis is present with periarterial fibrosis. Obliterative arteritis also exists. The vessel in the haversian canal running diagonally at the bottom of the picture no longer possesses a lumen. See figure 22. W. F., a woman aged 50, with a clinical diagnosis of otosclerosis; no. 1335 *A*.

changes appear to initiate these chemical reactions and to be the source of early disintegrative changes in the bone. The consideration of the

microchemical changes that occur within the vessels and in the tissue fluids, producing the dissolution of the normal structure of the bone, is beyond the scope of this paper. We here present the microscopic changes which we now believe to be a part of the otosclerotic process.

2. Hypervascularity. Vessels traversing the bone in these specimens are frequently supernumerary. In the sections the bone may appear to be

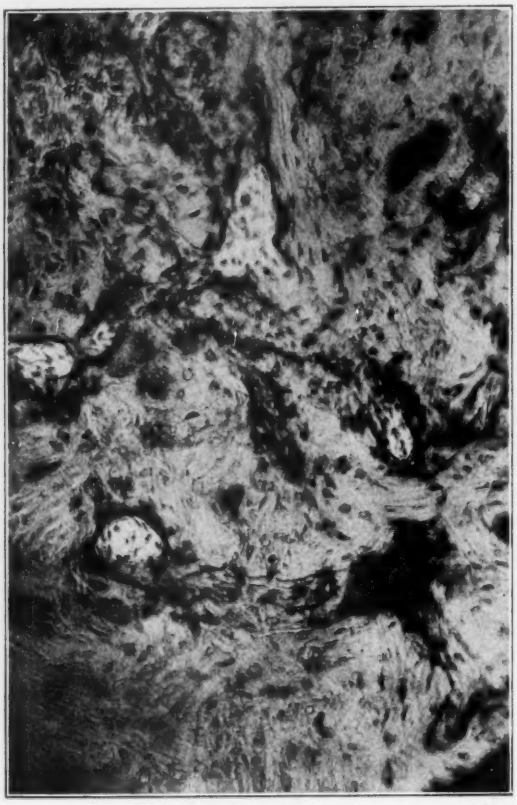

Fig. 17.—Pathologic bone showing a "seedy" appearance due to the enlargement of lacunas as the decalcifying tissue fluids spread from the haversian canals. W. M., aged 38; no. 548.

riddled with perforations. Common sites for such a condition are the neck of the malleus and the bone immediately underlying each articular surface. Multiplication of vessels in the initial stages of that

process is illustrated in figure 18. Tortuosity of vessels may expose the bone to a greater extent of vascular surface. Since, as already explained, tortuosity leads to congestion and stasis, the areas thus exposed may be subject to toxic fluids, with decalcifying processes resulting.

3. Decalcification. In the presence of congestion and stasis such as that illustrated in figures 12 and 13, stagnation of blood occurs. The consequent lack of ventilation and the described accumulation of gas in escaped globules both combine to make adjacent bone solvent.

Fig. 18.—Newly formed blood vessels penetrating the solid bone of the head of the malleus of a 20 year old man with a clinical diagnosis of otosclerosis. Congestion and stasis are present. Numerous minute clear globules are present within the lumen of the vessel. Others have escaped and are apparently enlarging as from the absorption of a gas (carbon dioxide?). No. 547.

4. Osteofibrosis. Following decalcification, the changes seen and the reactions noted in the tissue apparently depend on the individual constitution of the patient. Different patients react differently, according to their own constitution, once decalcification has been initiated. In figure 19 is seen an infarct on the superior surface of the incus. Fibrotic replacement of bone or osteofibrosis has occurred. The "ghosts" of the haversian canals are still apparent. In the high power view the release of

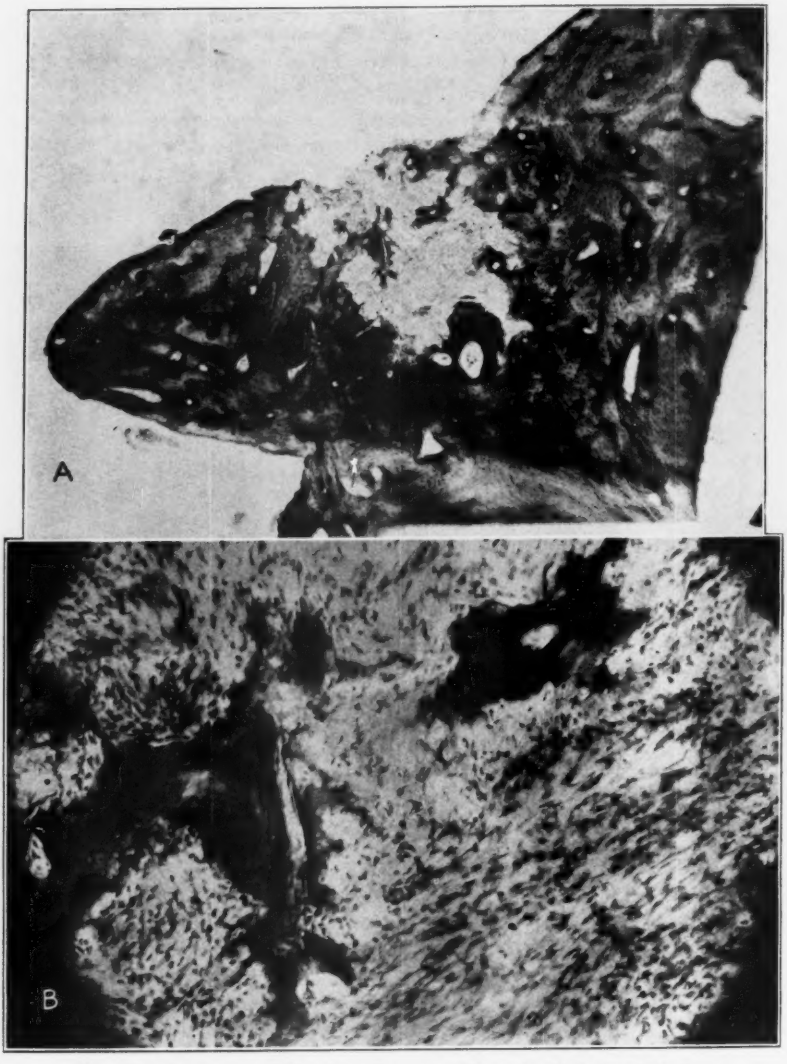

Fig. 19.—*A*, an area of fibrotic replacement of bone in the body of the incus. Note that the wall of a haversian canal has been completely eroded. W. F., aged 48; no. 2194. *B*, detail of *A* showing bone cells freed by the decalcifying process and taking part in fibrosis. Even the former lumen of the vessel has been invaded by fibroblasts.

the bone cells is evident and their fibroblastic activity may be observed. Figures 20 and 21 illustrate similar pathologic changes occurring in the incudes of younger persons, aged 18 and 36 years, respectively.

5. Osteoporosis. As stated, excessive numbers of vessels in bone produce numerous perforations. Thus the bone assumes a spongy

Fig. 20.—Osteofibrosis and malformation of the incus. W. M., aged 18; no. 1756.

appearance. If the blood supply to such an area is cut off, either from prolonged stricture or embolism, the soft tissue once supplied may become atrophic, necrotic and eventually disappear entirely. Figure 22 from the case illustrated in figure 16 shows an infarct in the incus just below

the articular surface. In *A* a constricted blood vessel is seen traversing an area of osteofibrosis. In *B* the area appears as a cyst which in *C* becomes a gouged-out area in the bony contour, which is quite irregular throughout.

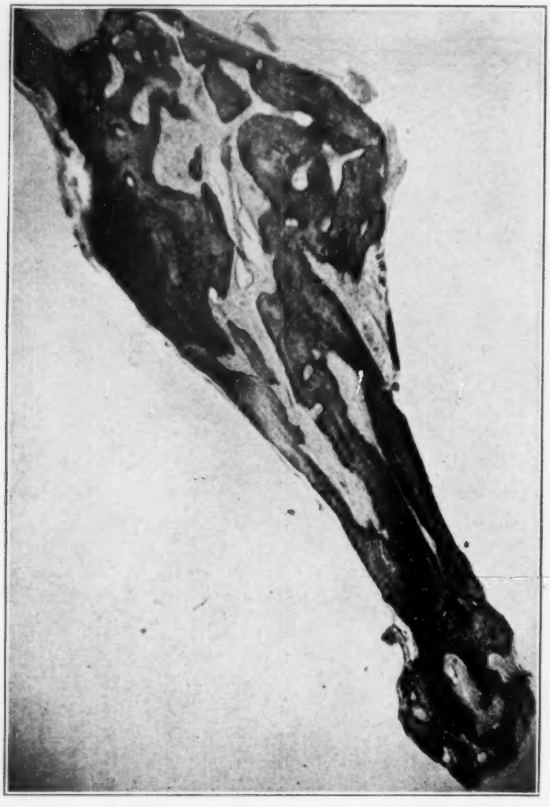

Fig. 21.—Osteofibrosis of the incus. W. F., aged 36; no. 1701.

In figure 23 we see a malleus and an incus, both of which are osteoporotic, and this pathologic condition is directly traceable to the obliterative endarteritis that occurred in the patient as illustrated in figure 14.

6. Malformations and exostoses. In persons who are suffering from a dyscrasia of calcium metabolism, malformations of bony structures

may occur with altered stresses and strains, or calcium may be deposited in bizarre locations. In the previous paper attention was called to the distorted shapes assumed by the incus and the malleus with altered muscular activity on the ossicular chain and altered activity from inability

Fig. 22 *A*.—An atrophic blood vessel surrounded by an area of fibrotic replacement of bone. See figure 16. W. F., aged 50; no. 1335 *B*.

to respond to sound vibration. That calcium is deposited in unusual locations is manifest in the exostoses of the disarticulated ossicles. Figure 24 illustrates this vividly, showing an exostosis on the superior surface of the incus. The line of demarcation between the normal contour and the new growth of bone is obvious. The growing bone is

highly vascular and contains large areas of fibrous tissue. It should be noted that the general staining pattern of this ossicle is irregular and that beneath the articular surface there is an extensive area of fibrotic replacement of bone. Figure 5 illustrates complete constriction of the

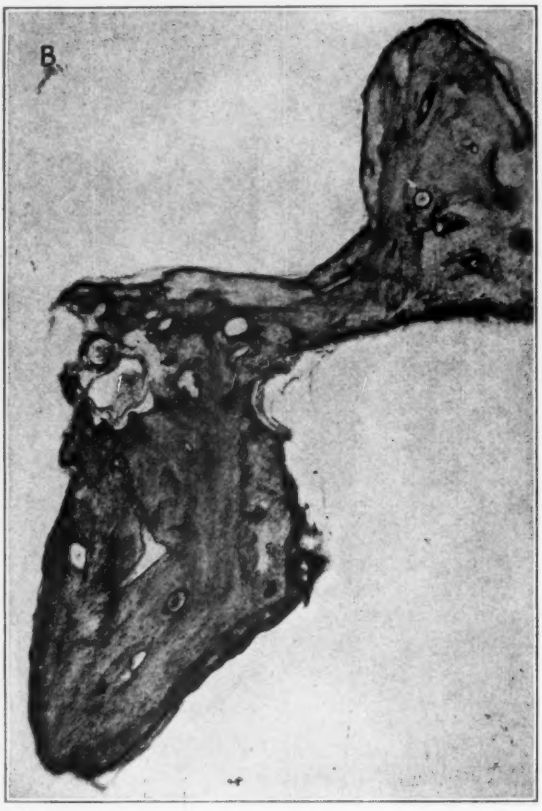

Fig. 22 *B*.—The fibrotic area gives way to a cystic space. W. F., aged 50; no. 1335 *C*.

vessel in the descending process of another incus. Calcium was not being normally distributed in this ossicle on account of occluded vessels; so the excess was deposited in abnormal locations.

An excellent example of the excesses to which such a pathologic process may go is well illustrated in figure 25 *A*, *B*, *C* and *D*. Here again it

is obvious that a pathologic state existed throughout this small bone. Areas of osteofibrosis and osteoporosis are numerous. Staining reactions are irregular.

7. Ankylosis. In pathologic states the deposition of calcium is uncontrolled as to location and amount. It may be deposited less abun-

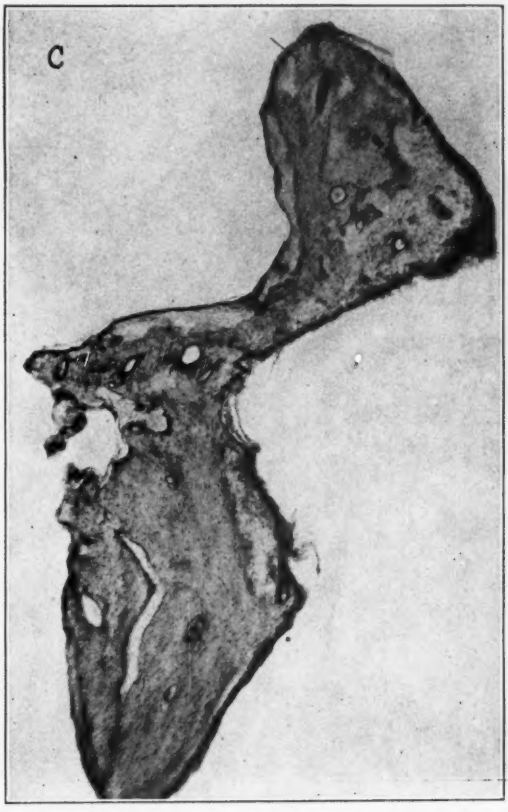

Fig. 22 *C.*—Superficially the area appears as osteoporosis, neither bone nor fibrous tissue being present. W. F., aged 50; no 1335 *D.*

dantly but equally insidiously as flecks in the ligaments of the joints or in the interarticular disks. In such a location it may not be long before calcium accumulates sufficiently to build a bridge of bone between the two ossicles. This results in ankylosis of the ossicles as illustrated in

figure 26 *A, B, C* and *D.*' As may be seen in this series of pictures, vascular channels may pass from one ossicle to the other. An area of osteofibrosis has also developed near the articular surface.

In figure 23 we see another example of ankylosis. The extent of the pathologic involvement can be fully appreciated only when the complete

Fig. 23.—Ankylosis of the malleoincudal joint in a case of bilateral otosclerosis. See figure 11 for detail of vascular pathologic process causing this condition. Osteoporosis is also present. W. F., aged 36; no 12. (Dr. MacCready made this specimen available to us.)

series of sections is studied. It is obvious, however, from this illustration that another type of bony change has occurred. Osteoporosis is also extensive. This condition, in turn, may be traced to the vascular state which has been illustrated in figure 14, a detail of figure 23.

Ossification in the area of ankylosis may be so extensive as to obliterate completely the interarticular disk. The incudostapedial joint shown in figure 27 *A* and *B* illustrates this condition. Only a small fragment of the interarticular cartilage is evident. An atrophic vessel

Fig. 24.—Note the exostosis or new bone formation on the superior surface of the incus. A sizable area of fibrotic replacement of bone is seen just below the articular surface. Erosion of articular cartilage has occurred superiorly. W. M., aged 22; no. 1333.

is seen in the head of the stapes. Indications are that this minute vessel may have been literally squeezed shut by the presence of globules producing edema on each side of it. Further pathologic changes are to be noted in the crura. Irregular contours are present, and, in addition

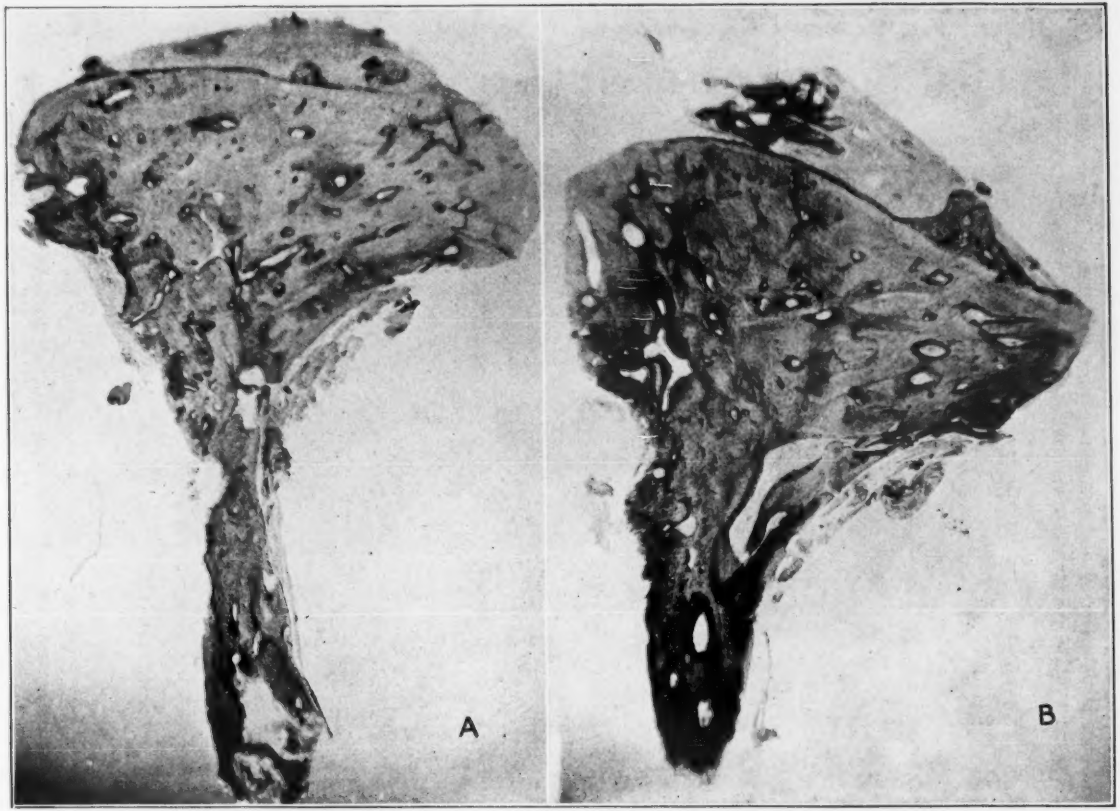

Fig. 25.—*A*, pathologic incus removed at a revisory operation. Note fragments of regenerated bone in the scar tissue lying above the incus. A small exostosis is also present. W. M., aged 40; no. 2060. *B*, another level of this incus, at which the exostosis is more extensive.

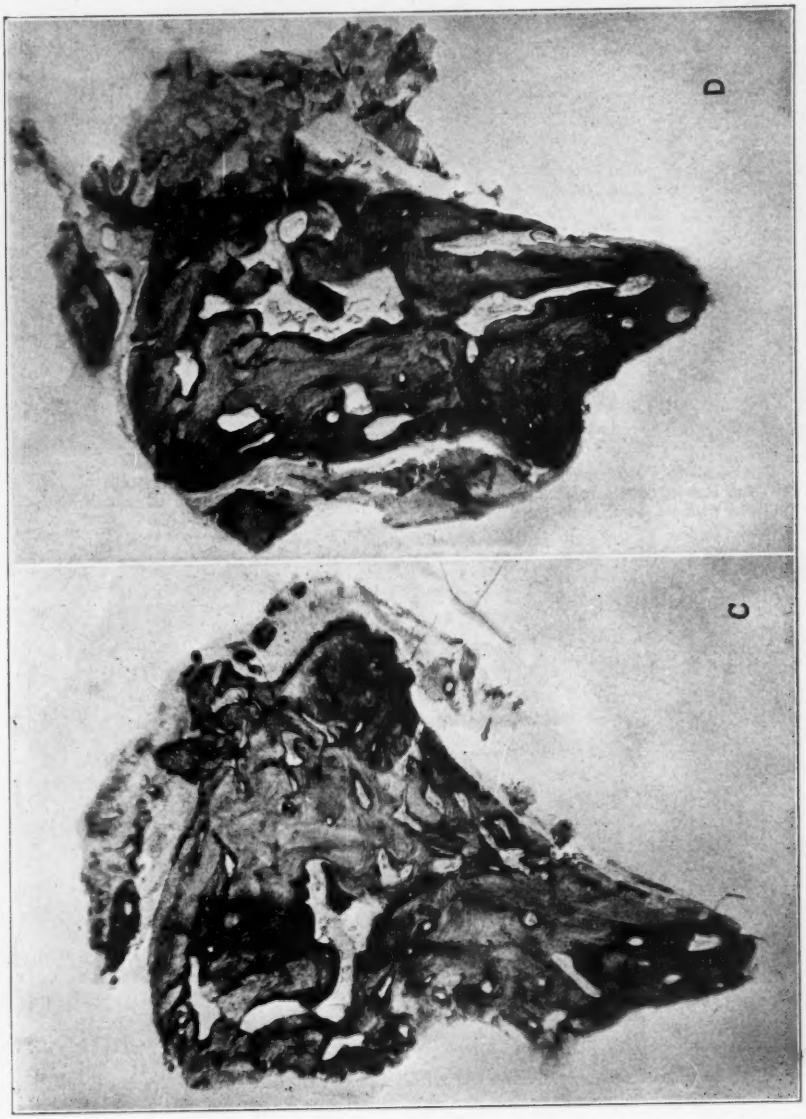

Fig. 25.—*C*, further development of the exostosis. *D*, malformation of the incus, due to the exostosis.

Fig. 26 *A*.—True bony ankylosis of the malleoincudal joint. Note the flecks of calcium in the interarticular disk and the area of fibrotic replacement of bone in the malleus, adjacent to the area of ankylosis. W. F., aged 30; no. 1784.

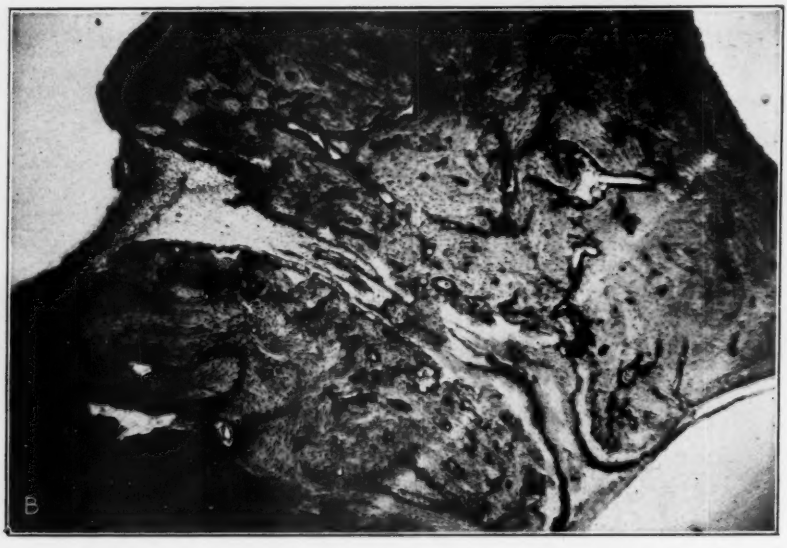

Fig. 26 *B*.—The ankylosed area as seen at a more superficial level. Note the area of fusion of articular cartilages and interarticular disk to the right of the ankylosed area. To the left of it flecks of calcium are seen in the interarticular disk. A minute blood vessel traverses the area of ankylosis.

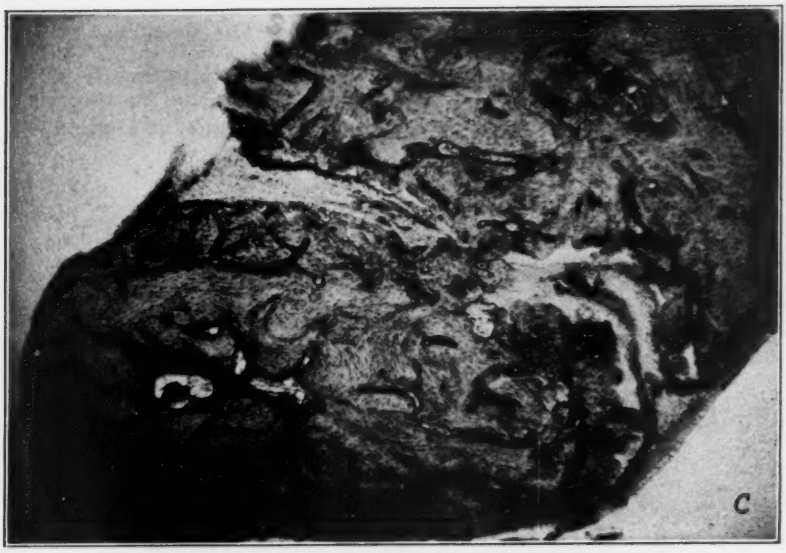

Fig. 26 *C*.—Just below the area of bony ankylosis an area of osteofibrosis may be seen. A sizable deposit of calcium occurs in the interarticular disk near the ligament of the joint.

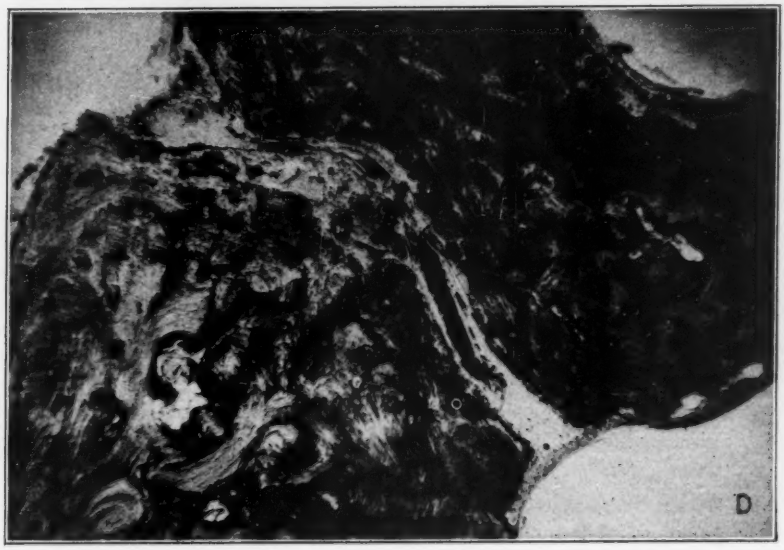

Fig 26 *D*.—The pathologic area as seen from a very superficial level. Note the extensive calcification of the interarticular disk and the fusion of the locally contiguous cartilaginous articular surfaces.

to an overgrowth of bone, osteoporosis has developed particularly in the posterior crus. This stapes appears to illustrate an otosclerotic condition quite typically. The pathologic area extends to a higher level on the crus than is commonly seen.

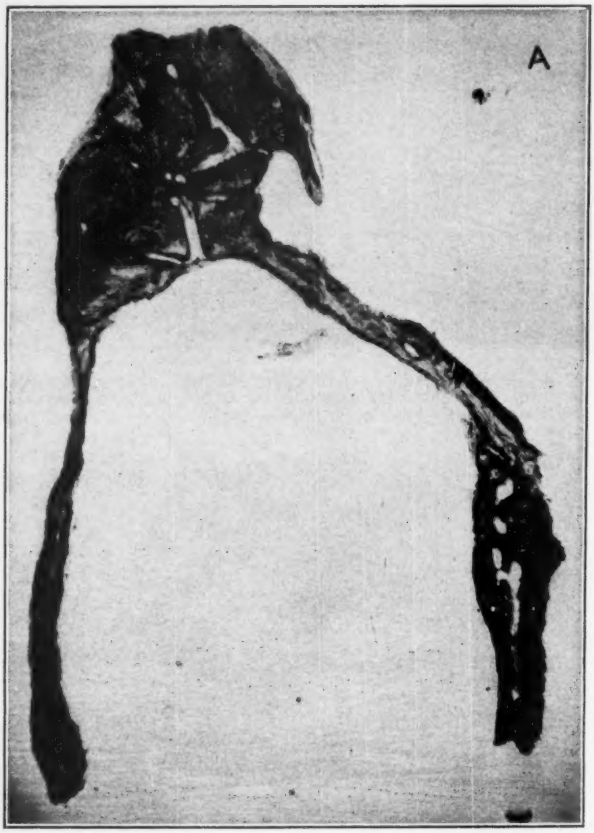

Fig. 27 *A*.—Otosclerosis of the stapes. Note the hypervascularity and the enlargement of the posterior crus. Ankylosis of the incudostapedial joint has also occurred. W. F., aged 44; no. 2202.

Figure 28 *A* and *B* illustrates another stapes of a 28 year old patient. Here the head shows extensive fibrotic replacement of bone, but such a condition has been observed as an anatomic variation. However, since

it is coupled with the condition of the lower end of the crus illustrated in figure 28 *B* and a clinical history of otosclerosis one is forced to recognize a pathologic condition. Here bone has been deposited extensively by accretion.

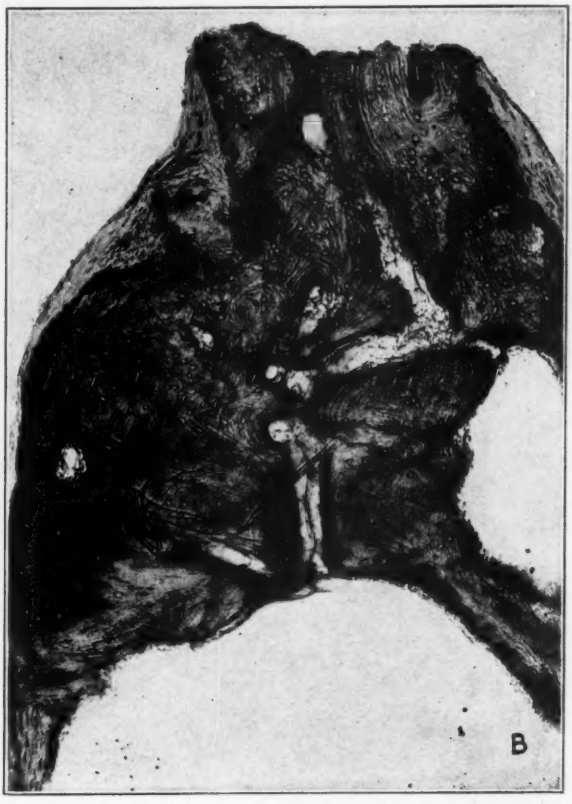

Fig. 27 *B*.—Detail showing ankylosis of the malleoincudal joint. Almost complete obliteration of the cartilage of the joint has occurred. The bone has been hypervascular, but the vessels are now atrophic. W. F., aged 44; no. 2202.

8. Ischemia. Attention has already been called to the irregularity of the staining of the bone in these specimens. Mention has been made of occurrence of complete stricture of vessels supplying a given region. If this constriction is long lasting or permanent, necrosis of bone cells may occur. This gives the microscopic picture of pale-staining areas of

Fig. 28.—*A*, fibrotic replacement of bone in the head of the stapes. *B*, deposition of excess bone by accretion on the lower part of the crus of stapes. W. M., aged 28; no. 1009.

Fig. 29.—Otosclerosis of the footplate of the stapes in a clinically diagnosed case of otosclerosis. No ankylosis was present. No otosclerosis of the otic capsule was seen at the oval window, but otosclerotic bone is present near the round window. W. F., aged 40; no. 5.

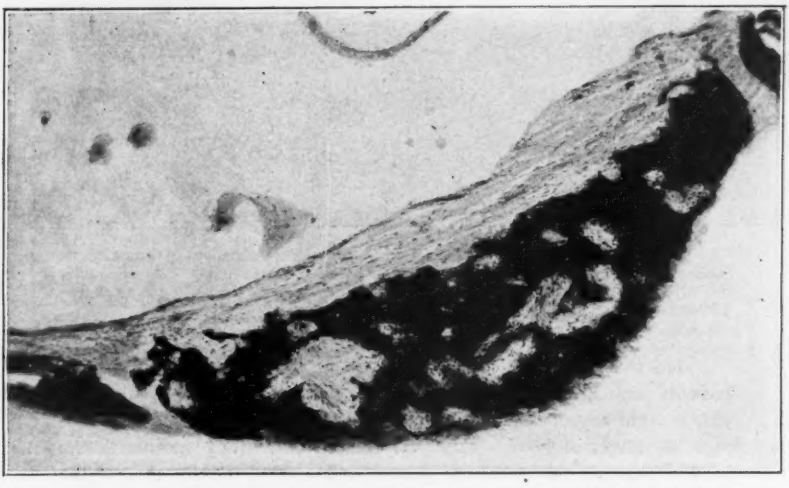

Fig. 30.—Detail of the stapedial footplate shown in figure 29 but taken at a slightly more anterior level and showing more extensive osteofibrosis. No evidence of ankylosis occurred at any level. W. F., aged 40; no. 5, left ear.

bone with lacunas devoid of bone cells. As stated, such an area may lie adjacent to an area with normal vascular supply and normal staining reactions. In bone lacking blood supply the bone cells die from lack of nourishment and eventually disappear entirely, leaving empty lacunas, but the bone itself will maintain its original contour. It is bone with an abundant blood supply or an excessive blood supply that is most apt to undergo change of form.

Fig. 31.—Congestion and stasis in the vessels of the malleus in otosclerosis observed in a postmortem specimen, unsuspected during life. W. M., an adult; no. 5178, left ear. (This photograph was made available to us by Washington University.)

SUMMARY

Thus it may be seen from this survey of 100 ossicles removed from patients with a clinical diagnosis of otosclerosis that one small ossicle may present any one or a combination of several types of known pathologic processes of bone. There appears to be nothing specific in the kind of reaction the individual patient will give to the presence of the disease.

Decalcification occurs without the presence of osteoclasts. Only in 2 cases were any osteoclasts observed in the tissue, but areas of decalcifi-

cation were numerous and extensive. Osteofibrosis, osteoporosis, hyperostosis and sclerosis may all occur. The cubic volume of the lesion when compared with the cubic volume of the skeletal anatomy of the body is minute. But because of the strategic location of the alteration the significance of its presence is great. The types of pathologic involve-

Fig. 32.—Fibrotic replacement of bone, congestion, stasis and cystic formation in the descending process of the incus in the case of otosclerosis illustrated in figure 31.

ment seen in these ossicles may also be seen within the ossicles in any proved series of cases of clinically diagnosed otosclerosis examined after autopsy. The lesion, however, is not circumscribed in the ossicles as it is in the otic capsule. (See figures 29 and 31.) This we believe to be due to the anatomic difference in the blood supply. The otic capsule receives

its blood supply from six different sources (perhaps more).[3] The ossicles receive their blood supply from one or at most two sources. In the otic capsule, with its abundant blood supply, it is possible for a circumscribed area supplied by a certain blood vessel undergoing pathologic changes to lie more or less completely surrounded by areas still receiving a normal supply of blood and therefore maintaining normal haversian systems and a normal histologic picture. The pathologic changes previously noted and the additional ones described in this paper are a part of the otosclerotic process.

We believe the vascular changes preceded the bone changes in the development of otosclerosis. We have therefore studied the blood vessels of the marrow spaces of the ossicles, the vessels of the mucosa when it was intact, the vessels of the head and neck of the malleus, the vessels of the short process of the incus and the vessels of the descending process of the incus. The condition of the walls of these vessels was examined. The histologic vascular content was also examined. The pathologic changes observed were found to lead in more or less logical and chronologic steps to the various types of bone disease described.

Changes of Vascular Content

1. Clear globules
2. Accumulating fragmented red blood cells
3. Sludge
4. Accumulating polymorphonuclear cells

Changes of Vascular Wall

1. Constriction of vessels the caliber of which is normally that of capillaries supplying bones elsewhere in the body
2. Tortuosity of vessels caused by:
   (*a*) Hypertrophy of endothelial cells
   (*b*) Varicosities
3. Periarteritis
4. Endarteritis

Changes of Bone

1. Excessive blue mantling
2. Hypervascularity
3. Decalcification
4. Osteofibrosis
5. Osteoporosis
6. Hyperostosis with malformations
7. Ankylosis of joints
8. Ischemia with empty lacunas

CONCLUDING COMMENT

A survey of pathologic changes seen in ossicles from 215 patients with a clinical diagnosis of otosclerosis leads us to believe that these

ossicles do show otosclerosis but not in the clearcut circumscribed form we had anticipated. Believing that in studying a large number of ossicles from these patients we might see progressive stages of the disease, we have here attempted to present the findings somewhat in a possible chronologic order.

We have demonstrated microscopic evidence of ankylosis in the incudostapedial and incudomalleolar joints of these ossicles. We believe the initial pathologic changes are within the blood stream.

Age Distribution of 100 Patients Whose Ossicles Have Been Histologically Examined

Between 12 and 20	20-29	30-39	40-49	50-59	60-69
18	29	36	43	52	63
	29	35	44	57	
	29	37	40	52	
	26	38	41	54	
	23	34	42	55	
	27	36	44	51	
	20	39	41	50	
	22	30	43	52	
	29	39	40		
	24	34	45		
	26	31	48		
	29	35	40		
	22	37	40		
	29	32	45		
	28	31	40		
	22	31	41		
	29	38	43		
	27	38	40		
	23	31	42		
	29	34	44		
	20	38	41		
	28	33	46		
	27	35	49		
		36	46		
		34	43		
		30	48		
		30	42		
		37	44		
		37	44		
		36	41		
		38	44		
		34	43		
		37	46		
			49		

We have taken into consideration the possibility that senescence plays a part in the production of the pathologic changes described. However, a survey of the decades of life represented in this series of patients precludes the possibility that geriatric conditions are a factor. We are dealing with a group of young persons, as may be seen in the table.

119 East Seventy-Fourth Street.

INHALATION OF PENICILLIN AND STREPTOMYCIN IN OFFICE PRACTICE

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WASHINGTON, D. C.

THE INHALATION treatment of respiratory diseases has been known for centuries. Steam has been used by itself or with various volatile drugs, such as camphor or menthol, for many years. Shortly after Koch announced his discovery of the tubercle bacillus, someone advocated the inhalation of a culture of another organism to kill the tubercle bacilli in the lung. Following World War I, inhalation of chlorine gas was in vogue as a remedy for a cold in the head or the chest. The more recent use of inhalations of epinephrine for relief of asthma is known to everyone, and today almost every medical journal has an article about the inhalation of penicillin or streptomycin for some respiratory or general condition. It is only natural that the antibacterial drugs should be used by inhalation, for with this method they are delivered directly to the site of infection and in a concentration sufficient to be effective against susceptible organisms.[1]

Today the problem is to determine the best method of inhalation, that is, the type of inhaler, the proper drug, the correct amount and the best diluent, if any.

In this paper I shall confine my remarks to the inhalation of nebulized penicillin and streptomycin as applied in office practice and in the home.

The inhalation of penicillin is now recognized as an effective treatment for various infections of the upper and lower respiratory tract. It is probably the simplest method of reaching all of the respiratory mucosa at one time with a drug that is effective against gram-positive bacteria. Diseases of the respiratory tract which are amenable to

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1. (*a*) Abramson, H. A.: Principles and Practice of Aerosol Therapy of the Lungs and Bronchi, Ann. Allergy **4**:440-456 (Nov.-Dec.) 1946. (*b*) Bryson, V.; Sansome, E., and Laskin, S.: Aerosolization of Penicillin Solutions, Science **100**: 33-35 (July) 1944. (*c*) Findeisen, W.: Ueber das Absetzen kleiner, in der Luft suspendierter Teilchen in der menschlichen Lungen bei der Atmung, Arch. f. d. ges. Physiol. **236**:367-379, 1935.

office inhalations of penicillin are rhinitis, sinusitis, nasopharyngitis, pharyngitis, laryngitis, bronchitis, bronchiectasis, infectious asthma and contact ulcer of the larynx. Not all patients are relieved or cured, but the method is effective enough to be included in one's armamentarium. There are many other respiratory conditions which are reduced by nebulized penicillin, such as pneumococcic or streptococcic pneumonia, pulmonary abscess and acute laryngotracheobronchitis, but these are not suitable for office treatment and are not discussed in this paper.

Fig. 1.—Pen-I-Sol nebulizer with face mask and rebreathing bag. It is used in all conditions other than sinusitis. A pressure machine can be used instead of the oxygen tank. (Made by the Oxygen Therapy Sales Company, Los Angeles.)

APPARATUS AND TECHNIC

The apparatus used in my office is simple. It consists of a nebulizer connected with a face mask and a rebreathing bag and a tank of oxygen which supplies the pressure to nebulize the penicillin (fig. 1). If sinusitis alone is to be treated, nasal tips and a suction-pressure apparatus (fig. 3) are used instead of the face mask and the rebreathing bag. The penicillin is dissolved to make a concentration of 150,000 units in 0.75 to 1 cc. of distilled water, isotonic solution of sodium chloride or phenylephrine hydrochloride N. N. R. and is placed in the nebulizer; when this is entirely used up 0.5 cc. of distilled water or the saline solution is put into the nebulizer and inhaled to utilize the penicillin which has adhered to the sides of the apparatus. As much as 250,000 to 300,000 units of penicillin may be used at one time; in such cases it is advisable to use up to 1.5 to 2 cc. of the diluent. Since penicillin G has been used, there is no discomfort with inhalation

of the larger number of units. Barach and co-workers[2] have devised a tablet containing 50,000 units of crystalline G penicillin. As many as 4 tablets will dissolve in 1 cc. of distilled or boiled water. The tablets are placed in the nebulizer and the water added to make the solution.

Treatments are given at the rate of one a day at least and in some cases two a day for several days, depending on the severity of the condition and the response of the patient. In most cases that are not chronic I advise the patient to take at least one treatment daily for six days, and not to be disappointed if the disease does not show any response until that number has been taken. Frequently patients will state after the third or fourth treatment that the condition is no better, but many of the same patients will volunteer the information that they feel better or cured after the fifth or sixth treatment. Chronic conditions may require many more than six treatments. The number must be judged by the patient's response or by the results of various examinations, including bacteriologic and roentgenologic examinations.

Organisms may become resistant to both penicillin and streptomycin, and this may be a reason why some infections do not respond to the inhalations. To overcome this, it is necessary to use a sufficiently large dose of the antibiotic in the beginning of the treatment. Ordinarily one need not consider the resistance of the organisms to penicillin, but at times the resistance to streptomycin becomes so great that it cannot be overcome even with very large doses.

OBSERVATIONS

The respiratory tract conditions most amenable to penicillin inhalations are those due to gram-positive bacteria, particularly the streptococci and pneumococci. Even a cold in the head will often respond to the inhalations. One does not use the inhalations routinely in treating colds, as these are self limited, but on trying the inhalations at the request of the patients, I have been agreeably surprised to find that some of them are relieved promptly of their symptoms. The nurse employed in my office can relieve herself of colds with one or two inhalations; the secretary gets no relief from them.

Infections of the nasopharynx, which are difficult to treat in some patients, often respond to the penicillin aerosol. This result is probably due to the effect of the penicillin on the infection of the nares or the sinuses which so often accompanies or precedes nasopharyngitis. The aerosol makes it unnecessary to swab the nasopharynx and also prevents the infection from spreading downward. If there is any secretion in the nose, this is removed by suction, and if necessary a decongestant such as phenylephrine hydrochloride or an ephedrine salt is sprayed into the nares. Taking one or two inhalations a day for about two days is usually sufficient.

2. Barach, A. L.; Rumsey, C. C., Jr.; Soroka, M., and Radar, D.: A Simplified Technic of Treating Sinusitis with Penicillin Aerosol, with Description of Foot Pump for Economical Nebulization of Penicillin and Other Therapeutic Aerosols, N. Y. State J. Med. **47**:1498-1500 (July 1) 1947.

Laryngitis is effectively treated with inhalations of penicillin, particularly in the acute stages. Often a speaker or a singer can get relief in one to four inhalations and is then able to speak or sing without difficulty. This type of treatment is most gratifying, as it is so simple as compared with any other method of instilling medicine into the larynx. In cases of acute laryngitis the patient is asked to take two treatments a day, in that way inhaling 300,000 units of penicillin. At the end of the second day or the fourth inhalation the hoarseness is usually so much improved that the patient can carry on in a normal voice.

The most striking results are seen in cases of cough due to acute bronchitis or occurring as an aftermath of that condition in which all other remedies have failed. Most of the patients have tried various cough medicines, inhalations of steam, drops in the nose, gargles, etc., without avail. Many are children with soggy nares, postnasal discharge, lymphoid tissue on the posterior part of the pharynx or enlarged lingual tonsils, with a constant hacking or even a productive cough. Treating cough in children is especially important to prevent bronchiectasis. After a few treatments with the nebulized penicillin these patients are greatly improved and many are entirely relieved. It may be necessary to give as many as ten treatments to get a good result, but most patients are improved in less than that number. It is suggested that patients with bronchitis or cough be examined by their physicians before the inhalations are started. This is no problem, as most of the patients have already been under treatment with their family physicians, who are glad to have them get the inhalations. Occasionally, a good result is obtained in the patient with a cough that has not cleared up under inhalations of penicillin by adding streptomycin, 0.3 Gm., to the penicillin or substituting streptomycin for the penicillin. Patients with cough are forbidden to smoke, as smoking can undo all the good effect of the inhalations. If there is any reason to believe that bronchiectasis is present, bronchograms are taken, and a bacteriologic examination of the coughed-up secretion is made. Often these bronchiectatic patients cough up phlegm containing gram-negative organisms which are more effectively treated with streptomycin than with penicillin.[3] In these cases I feel that the combination of penicillin and streptomycin is better than either one alone. When penicillin alone is used, the gram-negative organisms may not be affected. They in turn elaborate an enzyme, "penicillinase," which destroys some of the

3. Olsen, A. M.: Inhalational Therapy in Bronchiectasis, J. A. M. A. **134:** 947-952 (July 12) 1947.

bacteriostatic activity of penicillin.[4] By the addition of streptomycin to the penicillin aerosol the gram-negative organisms are affected, and this prevents the production of penicillinase. When possible I try to get these patients to take two treatments daily, to get 300,000 to 500,000 units of penicillin and 0.6 to 1 Gm. of streptomycin a day. Before the inhalation is given the bronchi are emptied by postural drainage. In these chronic cases the number of treatments depends on the response—usually a month is a fair length of time to give them before expecting any lasting results, but many of these patients will feel better and have less expectoration, with elimination of the odor, after even a few treatments. It is recommended that these inhalations be given preliminary to surgical treatment of bronchiectasis, in order to prevent complications during and after the operation. Inhalations are especially effective in bronchial conditions, as the drug is placed directly at the sites of infection. As Segal[5] said, "The clinical course is sufficient proof of the topical effectiveness of penicillin aerosol."

Patients with sinusitis may get much relief from inhalation of penicillin. This treatment is valuable in cases of generalized sinusitis complicating a head cold when the condition is too acute to warrant much if any nasal or sinus treatment. If the nose is clogged, a vasoconstrictor is sprayed into both nares, and if mucopus or pus is present, this is removed with a fine suction tube. The penicillin dissolved in 0.25 per cent phenylephrine hydrochloride solution instead of saline solution or water is then inhaled through nasal tips inserted tightly into the vestibules. The nasal apparatus [2] works by the alternating pressure and suction provided by a Venturi tube, and in this way the penicillin is inhaled directly into the sinuses. Most acute involvements will clear up after a few inhalations; the head feels clear, the headache is gone and the patient experiences a sense of well-being which is not achieved by the usual nasal treatments. Whether this is due to the effect of the oxygen or to the thorough shrinking with the phenylephrine or to the effect of the penicillin I do not know. Even purulent sinusitis may respond to the inhalations and thus obviate the necessity of irrigations of the sinuses, although a combination of irrigations and inhalations is at times necessary to clear up some of these purulent sinuses. One patient, a boy of 14 years, had generalized purulent sinusitis complicated with bilateral otitis media and mastoiditis.

4. Abraham, E. P., and Chain, E.: Enzyme from Bacteria Able to Destroy Penicillin, Nature, London **146**:837 (Dec. 28) 1940. Woodruff, H. B., and Foster, J. W.: Microbiological Aspects of Penicillin: VII. Bacterial Penicillinase, J. Bact. **49**:7-17 (Jan.) 1945.

5. Segal, M. S.: Inhalational Therapy in Treatment of Serious Respiratory Disease, New England J. Med. **229**:235-241 (Aug. 5) 1943; Medical Progress: Inhalation Therapy **230**:456 (April 13) 1944.

The left mastoid process had to be exenterated. It continued to drain after the operation. The tonsils and adenoids were removed, and the maxillary sinuses were irrigated on several occasions, with the recovery of much mucopus, but the nose remained full of mucopus and the ears discharged as before. Penicillin had been given by injection and sulfadiazine by mouth in large amounts without any apparent effect. Penicillin aerosol was then tried, and the secretion in the nose and the ears dried up within one week. Irrigation of the maxillary sinuses following one week of inhalations of penicillin showed them to be free of secretion.

Patients often come into the office with a self diagnosis of "sinus," and on examination not much is found to account for their stuffiness and headache or discharge. Allergy may account for the discomfort of some of these. Some of them actually have low grade sinusitis, manifested by a hyperplastic nasal membrane and clouding of the sinuses on transillumination, and have not responded to previous treatments of nose and sinus. In these cases inhalations of penicillin dissolved in 0.25 per cent phenylephrine hydrochloride solution give the patients the relief they seek, although in some not much change of the mucosa can be noted following the treatments. After about the fifth or sixth treatment they will usually volunteer the information that they feel relieved. In some the sense of smell returns, which had not been present for years.

In some patients chronic purulent sinusitis responds well to the inhalations, but the patients are told not to expect any miracle. Many of them have had different kinds of intranasal treatments and usually surgical interventions. They have long since given up hope of relief and refuse further manipulation of their noses. Any improvement experienced through inhalations is most welcome, and the ease with which the treatments are given is appreciated. It is necessary with some of these patients to aspirate the secretion and to spray in a decongestant before using the inhalations. Cultures are taken also, to determine the antibiotic to be used. Usually the organisms are gram positive and penicillin alone is effective, but if any gram-negative organisms are found or if there is no response to penicillin alone, streptomycin is added. Some patients who have had completely dark or cloudy sinuses on roentgenograms for years, with discharge, stuffiness, headache and other discomforts, are relieved symptomatically, and their sinuses show on roentgen examination a clearing which was hardly expected. With some of these patients it is necessary to irrigate the maxillary sinuses to remove the heavy secretion at some time during the course of their inhalations, but in others the suction-pressure inhalations alone are sufficient to empty the sinuses. In these the

results are particularly gratifying, as up to now no treatment has offered more than temporary relief.

Some asthmatic patients are helped by inhalations of penicillin, particularly those whose asthma is due to infection. The penicillin is dissolved in 1 per cent phenylephrine hydrochloride solution instead of water or saline solution, and this may also be a factor in giving relief. The effect of the penicillin on the infection of the paranasal sinuses which accompanies the infectious asthma in some of these patients probably plays an important part. Many of the patients have had surgical treatment which has failed to relieve their asthmatic symptoms, in part because it does not eradicate all the foci of infection. In these patients nebulized penicillin probably reaches the hidden areas of infection in the sinuses and also in the bronchial tree, and on the elimination of these the asthma is relieved.

As time goes on other conditions are treated, with gratifying results. The inhalations are now being used in cases of contact ulcer of the larynx with good effect. Silence alone is of value, but the recovery is discouragingly slow. By adding inhalations of penicillin to the treatment one is able to eliminate the infection not only in the ulcer but in the upper respiratory tract which may contribute to the prolongation of the condition. The patient also feels that the doctor is doing something active for him besides advising him to keep silent. This course was suggested to me by Dr. Samuel Crowe, of the Johns Hopkins University, and is proving to be successful.

Acute tonsillitis and pharyngitis can be reduced by inhalations of penicillin, either through the local effect or through the absorption of the pencillin into the blood stream. The results are difficult to determine, as other remedies are combined with the penicillin inhalations, and also the condition clears up in a short time under almost any kind of treatment. The inhalations help by removing any infection which may be present in the nose and the nasopharynx and which may contribute to the continuation of the condition of the throat.

The ease of administering the inhalations is also a factor in their favor. Patients practically give themselves the treatments. After the initial examination, if the doctor prescribes inhalations, the nurse adjusts the apparatus to the patient's face or nose, and after the penicillin aerosol has been inhaled, she adds 0.5 cc. of distilled water or saline solution to the nebulizer in order that the penicillin which clings to the inner surface of the apparatus may be utilized. When the patient returns for further inhalations, the nurse gives the treatment and the doctor needs to spend only a minute or two to see how the patient is getting along. If there is any secretion in the nose which should be aspirated, this is done, and a decongestant is sprayed in before the inhalations are begun. If there are any cutaneous irritations, such

as urticaria, or any asthmatic symptoms following any of the inhalations, the inhalations are either discontinued or another type of penicillin is used. Asthmatic attacks occur seldom but may be severe and are treated with antihistamine drugs, such as diphenhydramine hydrochloride (benadryl hydrochloride®) or tripelennamine hydrochloride (pyribenzamine® [N'-pyridyl-N'-benzyl-N-dimethylethylenediamine]). Irritation of the mouth has not occurred since a humidifier was attached to the oxygen tank. However, if one uses a nebulizer held between the lips—i. e., without a mask—it is advisable to rinse the mouth after the treatment. Penicillin urticaria can be treated with nicotinic acid, 35 mg. in 8 to 10 cc. of distilled water being injected intravenously.[6] Segal[7] suggested changing to another type of penicillin when these local reactions occur.

OTHER TYPES OF APPARATUS

There are several types of apparatus other than the pen-i-sol® nebulizer, with rebreathing bag and mask (fig. 1), made by the Oxygen Therapy Sales Company, Los Angeles, and the negative pressure sinus apparatus, made by the Inhalational Equipment Company, Inc., New York, which I use. McAuliffe and Mueller[8] have devised a suction-pressure machine which works with an electric motor. Nebulizers made by the Inhalational Equipment Company, Inc., the Oxygen Equipment Manufacturing Corporation and the Vaponefrin Company, as well as the DeVilbiss nebulizer No. 40 and the Parke, Davis & Company epinephrine inhaler are all satisfactory and are supposed to deliver particles between 0.5 and 2.5 microns in size. For home use there are several types of apparatus available: The Abbott Laboratories have devised a small plastic atomizer to be used with micronized penicillin dust instead of penicillin solution (fig. 5). A small cartridge ot 300,000 units of finely divided penicillin is inserted into the apparatus, and the penicillin dust is breathed in through either the mouth or the nose. Krasno[9] reported that several hundred patients had used this apparatus with good results and no side effects. However, several of my patients have had severe reactions after using this type of apparatus. Their noses became congested and the skin of the nose and lips red and swollen. They felt as if they had a severe cold in the head.

6. Service, W. C.: The Treatment of Penicillin Urticaria with Nicotinic Acid, Ann. Allergy **4**:397-398 (Sept.-Oct.) 1946.

7. Segal, M. S., and Ryder, C. M.: Penicillin Inhalation Therapy, New England J. Med. **236**:132-138 (Jan. 23) 1947.

8. McAuliffe, G. W., and Mueller, G. C.: Aerosol Penicillin Administered in Paranasal Sinusitis with Balanced Suction and Pressure, Arch. Otolaryng. **46**:67-71 (July) 1947.

9. Krasno, L. R., in discussion on Davis, D.: Inhalation of Penicillin and Streptomycin in Office Practice, Tr. Am. Acad. Ophth. **52**:273-282 (March-April) 1948.

Fig. 2.—Vaponefrin® plastic mask and plastic nebulizer without rebreathing bag. It is used in the same way as the apparatus shown in figure 1. (Made by the Vaponefrin Company, Upper Darby, Pa.)

Fig. 3.—Simplified negative pressure sinus unit. This is similar in action to the sinusillin® apparatus described in my previous paper (Davis, D.: Inhalation of Penicillin and Streptomycin in Office Practice, Tr. Am. Acad. Ophth. **52**:273-282 [March-April] 1948). (Made by the Inhalational Equipment Company, Inc., New York.)

Barach, Rumsey, Soroka and Radar [2] have recently added a foot pump apparatus to their negative pressure sinus apparatus. This apparatus replaces the oxygen tank and allows the inhalations to be used at home with less expense to the patient. The nebulizer is fitted with nose pieces and a metal Venturi tube, and there is no rebreathing bag. I do not believe that it will become popular.

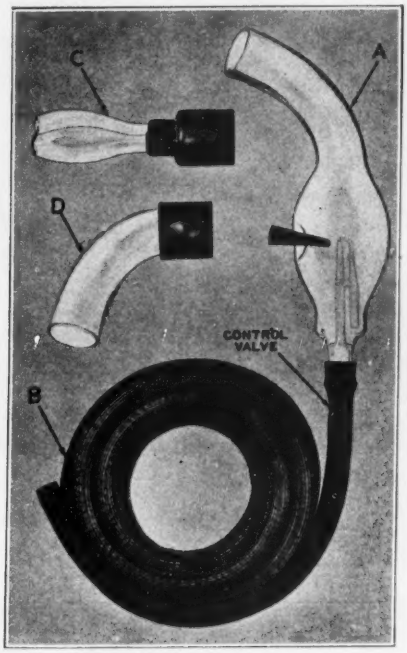

Fig. 4.—DeVilbiss no. 40 nebulizer for mouth (*A*) or nasal (*C*) inhalation. Instead of the long tube (*B*) which connects with an oxygen tank, a hand bulb can be attached directly to the nebulizer for home use. (*D*) is a glass tube which connects with (*A*) for use when the patient is lying in bed and cannot sit up.

Finke [10] has advocated the use of a bicycle pump to supply the air to nebulize the penicillin for home treatment. Prigal and associates [11] have devised a combined steam generator and nebulizer which can be used in the office or in the patient's home (fig. 6). Prigal stated

10. Finke, W.: Simplification of Penicillin Aerosol Therapy for Home Treatment, Am. Pract. **1**:643-644 (Aug.) 1947.

11. Prigal, S. J.; McGavack, T. H.; Speer, F. D., and Harris, R.: Aerosol Penicillin, J. A. M. A. **134**:932-938 (July 12) 1947.

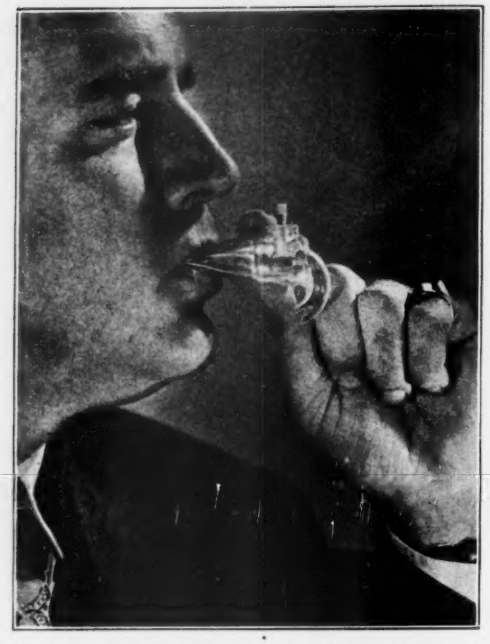

Fig. 5.—Plastic apparatus made by the Abbott Laboratories and introduced by Drs. Krasno and Karp. It may be used by mouth or nose. There are several plastic aerosol apparatuses now available, such as the Squibb dispolator® and the Upjohn inhalator.®

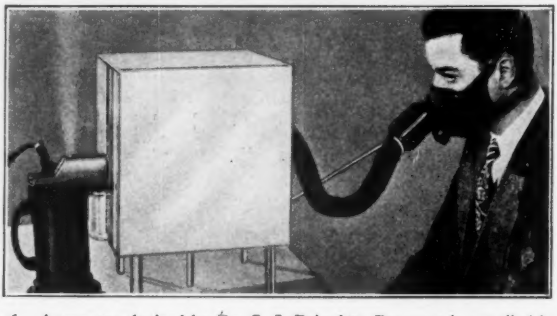

Fig. 6.—Apparatus devised by Dr. S. J. Prigal.[11] Pressure is supplied by steam.

that "satisfactory aerosols were readily obtained from solutions of theophylline ethylenediamine [aminophylline U. S. P.], epinephrine, sulfadiazine and penicillin—singly or in combination." He stated that he uses propylene glycol as a solvent for the penicillin and that "the use of a combined penicillin–propylene glycol aerosol under a tent provides a means of obtaining rapid and prolonged levels of penicillin in the blood." Senturia and Doubly [12] stated, however, that "propylene glycol completely inhibited the antibacterial action of penicillin and was incompatible with streptomycin" in vitro. Until this question is finally settled I have discontinued using propylene glycol as a solvent

Fig. 7.—Plastic hood that is useful in the treatment of infants and young children. (Made by the Oxygen Therapy Sales Company, Los Angeles.)

for penicillin and streptomycin and use either isotonic solution of sodium chloride, distilled water or phenylephrine hydrochloride.

For the treatment of infants and small children who cannot use the face mask or the suction-pressure apparatus there is a plastic hood which when tucked around the head and neck makes an airtight tent into which the penicillin aerosol can be piped (fig. 7). This works

12. Senturia, B. H., and Doubly, J. A.: Treatment of External Otitis: Use of Vehicles and Antibiotics in External Auditory Canal; in Vitro Studies, Laryngoscope **57**:633-656 (Oct.) 1947.

much like the oxygen tent used in hospitals but is smaller and can be used in the office or the home. Because of the short duration of the treatment, it is not necessary to cool the aerosol penicillin, and the patient soon learns to accept the treatment without much resistance.

Compressed air may be used instead of oxygen, and, according to some observers, there is practically no difference in the results. In my office compressed oxygen is still used, as this has proved satisfactory and gives the patient a feeling of well-being not experienced with compressed air.

No doubt many other drugs and antibiotics will be used by nebulization or are already being used, such as bacitracin, hydrogen peroxide,

Fig. 8.—Nebulizer with manual demand valve and rebreathing bag. This is made also without the rebreathing bag. Instead of the demand valve which saves oxygen, a Y tube may be used. The Y tube is open during expiration and closed during inspiration. (Made by the Inhalational Equipment Company, Inc., New York.)

chlorophyll, aminophylline and iodized oils. As better apparatus is devised and new drugs are discovered, aerosol therapy, I feel sure, will be even more firmly entrenched in the program of eradicating disease.

SUMMARY AND CONCLUSIONS

Inhalation therapy is recognized as an effective treatment for various infections of the upper and lower respiratory tracts. It is a simple method of applying drugs to the entire respiratory mucosal tract in sufficient concentration to be effective against bacteria.

The drugs most commonly used are penicillin and streptomycin. Bacitracin, hydrogen peroxide, urea peroxide, the sulfonamides, chlorophyll, aminophylline, iodized oil and others are also used effectively.

The apparatus consists of a nebulizer, which connects with a face mask and a rebreathing bag, and a tank of oxygen, which supplies the pressure. Compressed air can be used instead of the oxygen. For sinus treatments, nasal tips and a suction-pressure apparatus are used instead of the face mask and the rebreathing bag.

Inhalation treatment is effective in cases of acute or chronic sinusitis, nasopharyngitis, pharyngitis, tonsillitis, laryngitis, bronchitis, bronchiectasis, cough and infectious asthma. Even colds in the head frequently respond to the inhalation of penicillin.

In cases of infection of the respiratory tract due to gram-positive organisms, penicillin aerosol alone is effective. If gram-negative organisms are present, streptomycin, alone or combined with penicillin, is used. The inhaling of 150,000 units of penicillin once or twice a day for six days or less is usually sufficient to reduce most acute or subacute conditions. For chronic ones, such as bronchiectasis or chronic sinusitis, daily treatments may be necessary for thirty days or longer. If streptomycin is used, 0.3 to 0.5 Gm., alone or combined with penicillin, is given.

Treatments may be taken at home with any one of the various types of apparatus described.

Infants and small children can be treated through a plastic hood tucked around the head and neck.

1835 I Street, N. W.

ABSTRACT OF DISCUSSION

DR. ALVAN L. BARACH, New York: As Dr. Davis has outlined, there is evidently considerable value in the inhalation of penicillin mist in office practice. It is true that difficulties are encountered in interpreting the effect of pencillin aerosol under negative pressure in acute sinusitis. In a good many of the patients the conditions clear up under conservative measures, but in regard to those with chronic sinusitis an opportunity is afforded to test the effect of negative pressure in conjunction with penicillin aerosol in patients who have had histories of suppurative disease for five, ten or fifteen years.

I should like to show a few slides of people who have been treated over a period of years.

This is the picture of ethmoid sinusitis in a girl treated in a series at the Presbyterian Hospital in a study conducted by the nose and throat department, with Dr. Glass, and the department of medicine, with Dr. Pons and Dr. Garthwaite.

This patient not only obtained complete clearing away of a chronic nasal discharge but also, quite interestingly, was transformed from what appeared to be a behavior problem, a girl who seemed like a spoiled child, very alienating, into one of the most attractive, cooperative patients in the clinic.

She was given 200,000 units of penicillin once a day in a 1 per cent solution of an ephedrine salt. From our experience, three to four treatments per day are preferable.

A boy who had suffered from chronic sinusitis for fifteen years and whose sinuses had been irrigated many times, was hospitalized and after twelve days was free of the symptoms of purulent chronic sinusitis.

One of the interesting things is the disappearance of cough which often takes place in these cases, not caused by bronchitis or bronchiectasis; this is due to the treatment of the nasopharyngitis which, I think, is often present.

Dr. Samuel J. Prigal, New York: I agree with what was said by Dr. Davis and Dr. Barach concerning the value of penicillin aerosol in the treatment of respiratory infections. However, I have used a different method of therapy.

It was my belief that it was wasteful and costly to employ oxygen merely as a driving force; therefore, I sought a cheaper method of producing aerosols and began to use a steam generator, using the force of steam to create the aerosol. I have developed an apparatus which is efficient and does not necessitate repeated replacement of oxygen. With the combined steam generator and aerosolizer, one simply goes to the sink for a refill.

Having produced an aerosol which was inexpensive, I next faced the problem: how to make that aerosol last long enough to enable one to make good use of it. This was achieved by employing propylene glycol as a solvent for the penicillin.

Any aqueous aerosol which one may produce evaporates. The finer the aerosol the greater the surface and therefore the greater the evaporation. With this steam generator which Dr. Davis mentioned, using propylene glycol as a vehicle in which various medicaments are dissolved, I can produce an aerosol which will last an hour, if desired. It enables me to treat patients under a tent or in a small chamber at home, i. e., the bathroom. I like the bathroom treatment for children particularly, because it frequently enables me to treat simultaneously a mother or a father who may act as a carrier, along with the child. I select for treatment the parent who on examination shows evidence of being responsible for repeated reinfection of the child.

The next problem was that of confining the aerosol in such a way as to give the patient the maximum use of it. I confined the patient in the bathroom or in a small chamber and blew the aerosol in. I have also used tents, but the most effective and efficient method is the one in which I use what I call a breathing box. The aerosol dissolved in propylene glycol is blown into this box, and the patient inhales from this box by means of tubing and a mask. With the use of either the tent or this breathing box, it has been possible to demonstrate penicillin in the blood in high concentration for six hours. It has enabled me to use this method as an adjunct to the giving of continuous penicillin therapy for systemic disease. It also means for me that I do not have to treat my patients every two or three hours. Severely ill patients are treated twice daily, rarely more times; most patients receive only one inhalation daily .

Steam in itself, in addition to penicillin or streptomycin, has such value that it is of special merit to the practitioner of otolaryngology.

Dr. David Davis, Washington, D. C.: I should like to show a few slides to illustrate the apparatus I talked about. I might mention some of the apparatus which can be used at home. Dr. Barach has devised a foot pump to supply the power for the nebulizer, and Dr. Fink uses a bicycle pump. I have had no experience with these. I often advise home inhalations in addition to the inhalations given in the office and think I get better results.

Dr. Barach's apparatus consists of a nebulizer with nasal tips. It has a Venturi tube attached to rubber tubing leading from the oxygen tank in order to get alternate pressure and suction. When the Venturi tube is closed, oxygen goes through the nebulizer and the patient breathes it through his nose. When the patient removes his finger from the tube, allowing it to open, the oxygen escapes, leaving a vacuum in the apparatus. The patient swallows at the same time, the soft palate closes the nasopharynx and this creates a vacuum in the nose and sinuses. Then when the patient puts his finger on the Venturi tube and closes it, penicillin and oxygen are inhaled into the nose and the sinuses. This procedure is repeated many times during the treatment and is effective in the treatment of infected sinuses.

A DeVilbiss nebulizer can be used at home with a hard bulb or in the office with compressed air or oxygen. There are several small compression machines suitable for home or office use.

Dr. Prigal uses a plastic box about 12 inches (30.5 cm.) in all dimensions with a nebulizer inserted into its back wall and the face mask connected with its front wall. Pressure is supplied by a steam kettle heated electrically and attached to the nebulizer. The aerosol is nebulized into the box and is breathed in by the patient through the face mask.

Drs. Krasno and Mary Karp have been demonstrating a small plastic apparatus with either mouth or nose attachments. A cartridge of 100,000 units of penicillin is inserted, and as the patient breathes in, a little metal ball hits the cartridge and shakes a little penicillin dust into the floor of the apparatus. This is inhaled into either the nose or the mouth. On exhalation the apparatus is removed from the nose or the mouth so as not to blow moist air onto the cartridge of penicillin. A little sulfanilamide powder is mixed with the penicillin in the cartridge to make easier flowing. The main objection of my patients who have used this apparatus is that the penicillin does not shake out of the cartridge at times. It is a clever apparatus.

A little plastic canopy can be used with infants and small children. It fits over the head and has a special nebulizer which attaches to the top. The patient lies down for the treatment, and this can be used in office, hospital or home.

I wish to mention again the boy with sinusitis and bilateral mastoiditis to confirm what Dr. Barach said about the value of penicillin aerosol in chronic sinusitis. This boy, 14 years of age, had pansinusitis and bilateral otitis media; one side had to be operated on for acute mastoiditis. The ears continued to drain in spite of nasal treatment, removal of adenoids and tonsils and injections of penicillin. After one week of inhalations of penicillin, without any other therapy, the condition cleared up entirely. It sounds a little spectacular, but many patients have responded in just such a manner.

ROLE OF COMPENSATORY HYPERTROPHY AND SIMPLE ATROPHY IN INTRANASAL SURGERY

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THE PURPOSE of this paper is to report a method of changing the aerodynamics of the nose through intranasal surgical procedures, in a specific manner, so as to initiate spontaneous reactions of the nasal tissue and thus release the compensatory forces of hypertrophy and simple atrophy which will finally restore normal physiologic processes. These forces are basic nasal compensatory mechanisms and are essential for physiologic restoration and control. The surgical technic described is based on the fundamental laws of the aerodynamic physics of the nose, which is a bilaterally symmetric organ dependent on these laws for physiologic activity. The cases presented were selected from those of a diversified group of patients of different ages to demonstrate that the spontaneous reactions initiated can be controlled over a period of time until the normal physiology is reestablished.

A brief review of the accepted functions and anatomic structure of the nose will serve as a basis for describing the surgical methods used by me in actual practice. First, the functions are considered, and then the structure on which these functions are superimposed to form the fundamental physiologic and anatomic pattern of the nasal compensatory area involved.

The functions of the nose are classified, usually, as respiratory, phonatory, olfactory, gustatory, and also ventilatory, which includes the ventilation of the nasal accessory sinuses and eustachian tubes. Maintenance of organic structure may be added as another function according to Haldane,[1] who has stated: "The assumption of the existence of organic regulation to both structure and function is absolutely fundamental."

Generally, from a surgical point of view, it is believed that two of the most important factors contributing to nasal health and comfort are adequate ventilation and drainage. However, after surgical intervention has established what appears to be adequate ventilation and drainage, numerous symptoms remain, and not infrequently others have developed.

1. Haldane, J. S.: The New Physiology and Other Addresses, Philadelphia, J. B. Lippincott Company, 1919, p. 89.

Consequently, accompanying any basic change in nasal structure there must be a corresponding change in function. As Machs says, "In every symmetrical system every deformation that tends to destroy the symmetry is complemented by an equal and opposite deformation that tends to restore it." [2] Proetz has pointed out that "in the nose, structure plays a fundamental part in the proper maintenance of physiologic air channels, streams and pressures, in the humidification of the air and in the conditioning of the nose itself." [3]

The anatomic structure of the nose is characterized by bilateral symmetry and consists of two nasal chambers bounded by a roof, a floor, a single mesial wall (septum nasi) and two lateral walls; the vestibules lined with mucous membrane, and three turbinate bodies covered with mucous membrane located on each lateral wall. "The two nasal chambers are more or less regular spaces, triangular in shape, situated on either side of the middle line of the face, extending from the base of the cranium to the roof of the mouth, and separated from each other by a thin vertical septum." [4] They communicate with the frontal, ethmoid, sphenoid and maxillary sinuses, and for all practical purposes are the continuation of the eustachian tubes. The vestibule is just within the anterior naris and is bounded on the median side by the soft tissues of the nose. The superior, middle and inferior turbinate bodies are covered with mucous membrane containing venous plexuses, known as "swell bodies," with the inferior turbinate bodies containing the larger number. The turbinate bodies are nearly parallel and approximately an equal distance from each other, subdividing the nasal passages into three channels called superior, middle and inferior meatuses.

The nasal septum consists mainly of the anterior triangular cartilage and the perpendicular plate of the ethmoid and the vomer, which are bony in structure. It is covered on both sides with mucous membrane and not only divides the two nasal chambers but acts as a rigid wall to make definite spacing when the turbinate bodies expand or contract to narrow or enlarge the chambers according to the atmospheric changes. The importance of the rigidity of this structural pattern has been emphasized by Pratt,[4] who stated: "If we are to obtain the utmost use of the nose, the septum must be straight, thin and in the middle line, and the turbinates of normal size and in place, thus an equal amount of work, in modifying the air as it passes through, is performed by the two nasal chambers" (fig. 1).

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2. Mach, E.: Science of Mechanics, translated by T. J. McCormack, Chicago, Open Court Publishing Company, 1902, p. 395.

3. Proetz, A. W.: Essays on the Applied Physiology of the Nose, St. Louis, Annals Pub. Co., 1941, p. 30.

4. Pratt, F. J., and Pratt, J. A.: Intranasal Surgery, Philadelphia, F. A. Davis Company, 1924.

When the bilateral symmetry of the nose is disturbed—that is, when for some reason the air flow is not equal between the two sides—a spontaneous biophysical reaction occurs which tends to restore the aerodynamic balance and equalizes the air flow between the two nasal chambers. Aerodynamically, the nose itself responds to any interference with its functioning as a balanced unit. This physiologic response may not provide better ventilation or better drainage, but always tends to maintain integrity of structure, or the aerodynamic balance of a bilateral structure.

Thus when the septum becomes deviated in any degree to either side, the air flow on the deviated, or convex, side is reduced by reason of the narrower space and the air flow on the opposite side is greater on account of the wider space on this side. Nature then proceeds to reduce the

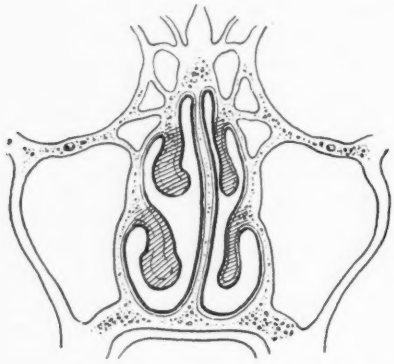

Fig. 1.—Diagrammatic cross section of the normal nose.

lumen on the wider concave side by enlarging the tubinate bodies and the other structures in an effort to restore the aerodynamic balance through so-called compensatory hypertrophy or "space-filling" reaction of the turbinates; at the same time a limited amount of space is gained on the convex side through simple atrophy or "shrinking" reaction (fig. 2). Compensatory hypertrophy and atrophy in the nose have been recognized and described by Pratt,[5] Jackson and Coates [6] and Morrison.[7]

5. "Deflected septa and 'spurs' on the septum make the nasal chambers unequal in size so that more air passes through one side than the other. Nature, in attempting to equalize the air space enlarges the turbinates by hypertrophies or hyperplasia on the concave side of the deflection. Large volumes of air, together with the dust and dirt passing through the concave side, create a congestion in the turbinates on this side; which, after a time, produce a hypertrophy

These reactions of "space-filling" and "shrinking" can be understood in relation to maintaining integrity of structure, a basic reaction of the organ itself; but they are not easily explained as a therapeutic measure because of the limitation of spatial capacity, which makes better ventilation and drainage more difficult.

The surgical technic described is based on the assumption that the physiologic reactions of compensatory hypertrophy and simple atrophy are basic organic forces which can be partially controlled and directed over a period of time. The control of these organic forces, initiated through intranasal surgical procedures in the compensatory area of the nose, is established by artificial changes in the aerodynamic balance of the nose because, according to Thompson, "no organic form exists except as is in conformity with physical and mathematical laws." [8]

Formerly, in a surgical case of deviated or obstructive septum with compensatory changes of the turbinate bodies and surrounding structures, either the submucous resection operation was used or the enlarged middle turbinate body was partly or totally removed (fig. 3). The submucous resection operation included removal of the greater portion of the cartilaginous and bony partition, leaving only the mucous membrane or "flap" to separate the two nasal passages. This residual partition is not rigid but is very much like a flexible diaphragm susceptible to movement back and forth by the air currents on either side in the process of breathing (fig. 4). With compensatory hypertrophy or thickening of the tissue and other structures in one side of the nose and with simple atrophy or thinning on the opposite side, this flexible partition moves to either side to accommodate the air flow, and, consequently, the nose is unable to control the air currents aerodynamically

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of the turbinates and so narrows the space. (It is a compensatory hypertrophy.) On the convex side the deflected septum presses on the turbinates, and nature tries to readjust the turbinates to make an adequate air space. As only a small volume of air can pass through, very little irritation is produced. The mucous membrane is thinned, which means a lessened blood supply, and in some cases even the bony part of the turbinate is reduced in size. (It is a compensatory atrophy.)" (Pratt and Pratt,[4] pp. 39-40.)

6. "Septal spurs and deviations are frequently accompanied by hypertrophy of the turbinal tissue in the wider naris. This is a compensatory hypertrophy and often is not pathologic." (hypertrophic rhinitis). (Jackson, C.; Coates, G. M. and Jackson, C. L.: Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 49).

7. "Hypertrophy of the mucosa and bone of the turbinates is seen with deviations of the septum of long standing; the middle and inferior turbinates on the wide side of the nose enlarge to compensate for the abnormal width of the air passage" (chronic rhinitis). (Morrison, W. W.: Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1938, p. 89).

8. Clark, W. E. Le G., and Medawar, P. B.: Essays on Growth and Form Presented to D'Arcy Wentworth Thompson, London, Oxford University Press, 1945, pp. 15-16.

in order to utilize this compensatory mechanism, or primary basic reaction. The natural organic regulation of the nose being no longer operative or effective, a spontaneous physiologic adaptation or adjustment which may be "advantageous but not designed" is initiated as a secondary basic reaction, becoming responsible for many of the pathologic variations in chronic nasal symptomatology.

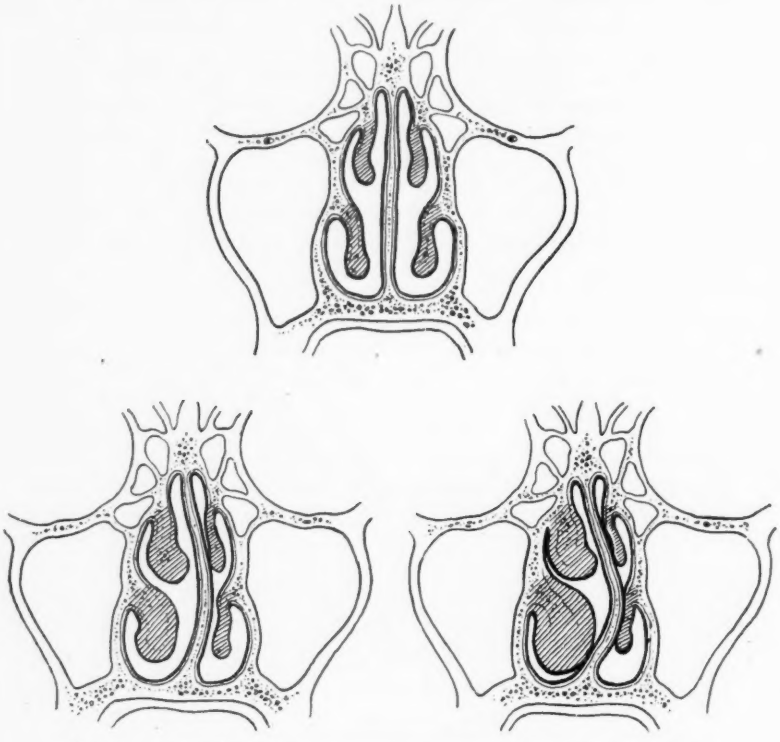

Fig. 2.—Progressive stages of compensatory changes occurring in deviation of the septum.

When the enlarged middle (or compensatory) turbinate body is partially or completely removed, the inferior turbinate body and the remaining structures continue the compensatory reaction by thickening and filling in most of the space previously occupied by the middle turbinate body (fig. 5).

The following corrective surgical technic, which I developed, is based on a biomechanical approach and requires that the maximum

structural rigidity of the septum be preserved. The septum must be moved over as nearly intact as possible into the concave side for the purpose of crowding the hypertrophied structures. This procedure is accomplished by separating part of the septum along the floor of the nasal chambers (from before backward), so that it can be mobilized

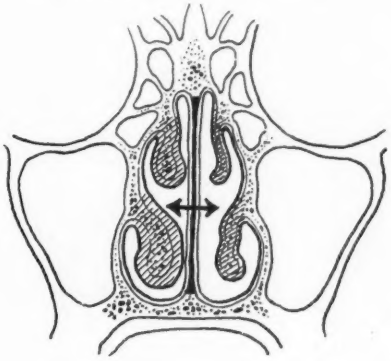

Fig. 3.—Diagrammatic cross section of deviated septum with compensatory changes in the turbinate bodies.

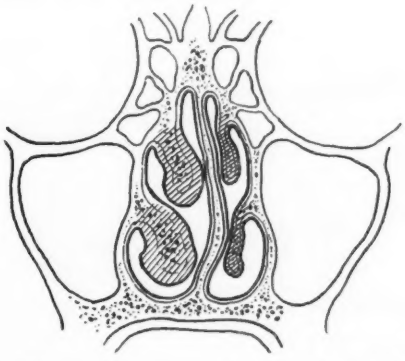

Fig. 4.—Diagrammatic cross section of a submucous "flap."

and forced over toward the nasal midline (figs. 6 and 7). A "submucous" incision is made as near the base of the septum as feasible, and the minimum amount of cartilaginous and bony structure necessary to effect mobilization and realinement is removed. The septum, now somewhat movable at its base, is fractured and forced toward the nasal midline with the "special" elevator illustrated in figure 8. The enlarged

and hypertrophied inferior turbinate body, present in most cases, should be fractured outward before one proceeds with the straightening of the septum (figs. 9 and 10).

A Simpson splint, of suitable size and shape, covered with dental wax, is inserted to hold the structures in their new position. Approxi-

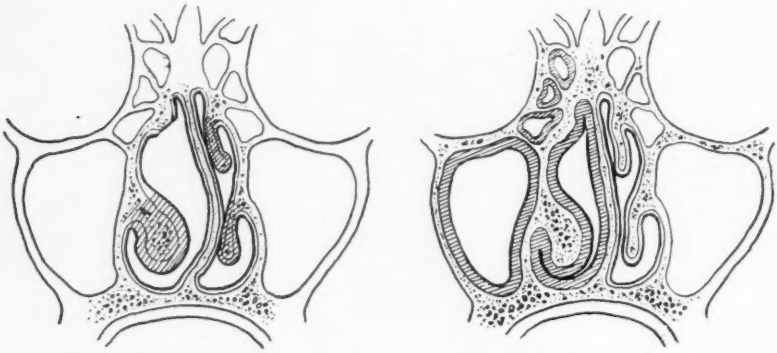

Fig. 5.—Diagrammatic cross sections showing (*left*) the middle turbinate body removed and (*right*) compensatory changes in the inferior turbinate body and other structures.

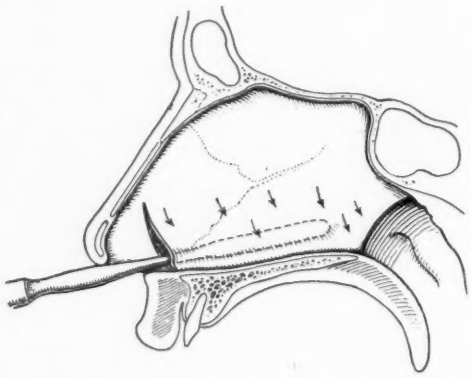

Fig. 6.—Elevation of the membrane along the floor with the Freer elevator.

mately twenty-four hours later the splint is removed; no packing is necessary. Some modification of these procedures must be considered in each individual case because of variations in anatomic contour. In actual practice the compensatory area of each nose must be redesigned

according to a definite long range plan in order to obtain permanent results.

In a typical case, after the operation or manipulation described, the septum should be in position in the nasal midline, the bilateral symmetry partly restored and the air flow controlled so that it can approach equalization on the two sides. A reduction of the thickened tissues and

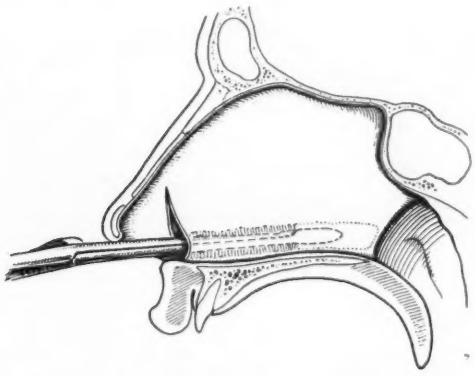

Fig. 7.—Release of the septum along the floor with a bone forceps.

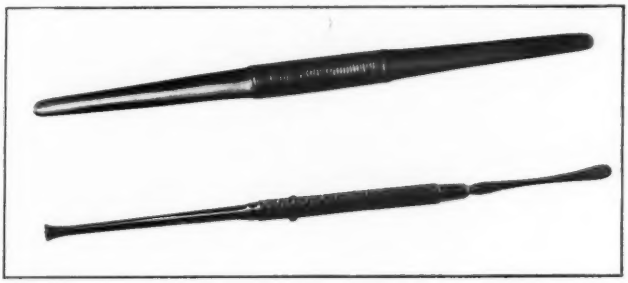

Fig. 8.—"Special" elevator elevator above; Freer elevator below.

other structures on the concave side now begins to take place; the thinned-out tissues of the convex side begin to be restored to normal thickness and at the same time normal physiologic functioning is being reestablished. This result has been accomplished by making "biomechanical" structural changes and by using the forces of compensatory hypertrophy and simple atrophy but in exactly the reverse order from that used by nature before the operation (fig. 11). These forces and

reactions, which have been initiated surgically, are self limited and follow a definite biophysical pattern. The time required for clinical recovery varies from a few days to several weeks. This is followed by the gradual return of the tissues to normal, which may continue over a period of

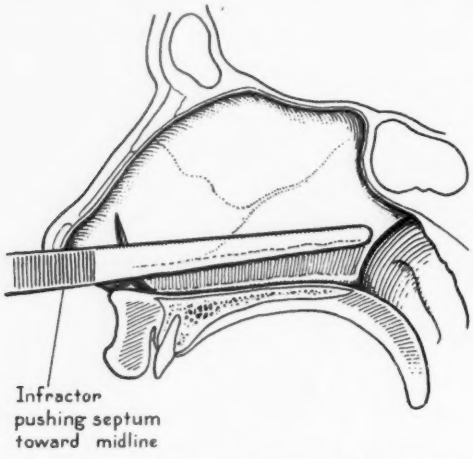

Fig. 9.—Use of elevator in realinement of the septum.

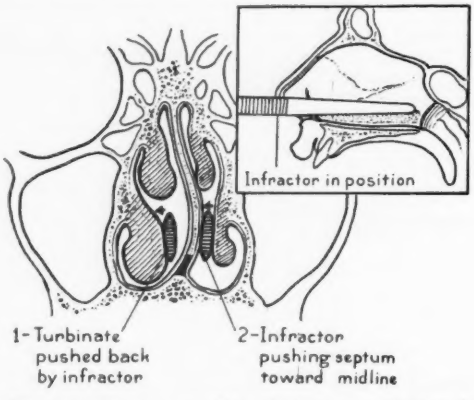

Fig. 10.—Diagrammatic cross section showing the elevator fracturing the septum and turbinate body.

months to several years, depending on each individual case. The application of these principles to chronic nasal obstruction in any age group presents new aspects in the field of preventive medicine.

The surgical technic described was used in the following selected types of cases: (1) systemic absorption; (2) chronic "sinus" headache; (3) reflex referred pain; (4) systemic absorption with chronic "sinus" headache; (5) allergic type with hay fever and asthma; (6) narrow and obstructive nasal passages, in (*A*) a child and (*B*) an adult; (7) deafness, catarrhal type; (8) deafness, combined type, and (9) deafness, purulent otitis media type. A tabulation of reported cases as well

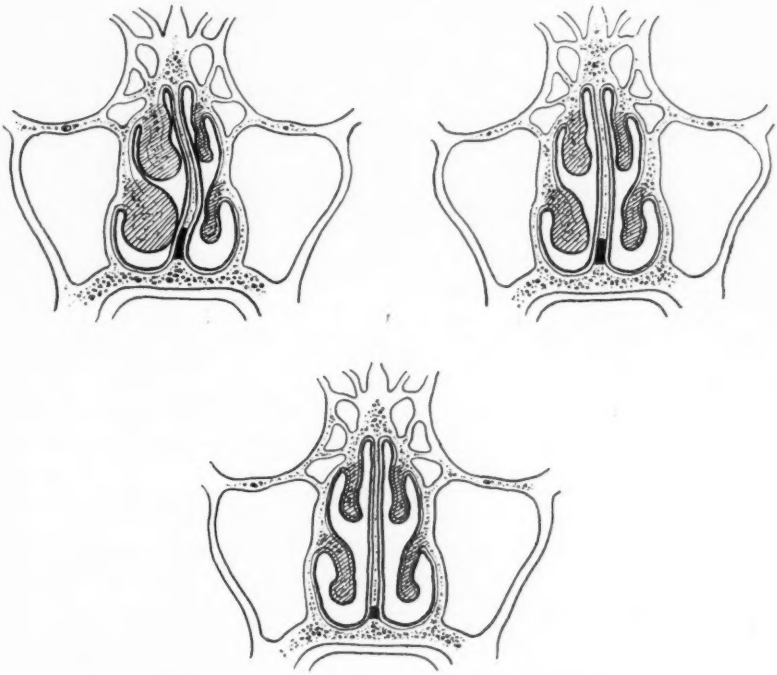

Fig. 11.—Diagrammatic cross section of typical deviated septum with compensatory changes in turbinate bodies, showing stages of septal realinement.

as a brief summary of each case is presented, including a description of the surgical technic.

REPORT OF CASES

CASE 1.—*Systemic absorption type; chronic rhinosinusitis with polypoid degeneration and deviated septum with spur formation.*

H. T. R., a man aged 39, was first seen Feb. 6, 1939, with chief complaints of generalized headache, pain in the regions of the frontal and maxillary sinuses, profuse nasal discharge, difficulty in breathing through the nose, sore throat, with

pain on swallowing, hoarseness, cough and general malaise. He gave a history of frequent attacks of similar nature, usually lasting six to eight weeks and more frequent in winter, for a period of years; general fatigue, poor appetite and rheumatic-like pain in the back and shoulder; he was 15 pounds (6.8 Kg.) underweight and both mentally and physically sluggish. He had been given a great deal of "sinus treatment," and a Caldwell-Luc operation had been advised.

Summary of Reported Cases

Case No.	Type	Sex and Age	Period of Observation	Physical Findings	Date of Operation	Result
1	Chronic rhino-sinusitis	Male, 39	2/ 6/39 to 5/15/47	Deviated septum, spur formation, polypoid degeneration, compensatory changes	6/30/39	6/30/40: Weight normal, free from nasal symptoms, tissues normal in color and general appearance to 5/15/47
2	Chronic "sinus" headache	Female, 32	2/26/44 to 3/15/47	Deviated septum, spur formation, compensatory changes	5/ 1/44	6/1/44: Free from symptoms, no recurrence to 3/15/47
3	Chronic rhino-sinusitis; referred pain	Female, 39	1/17/44 to 12/22/46	Deviated septum, spur formation, compensatory changes, generalized hypertrophy	1/18/44	5/18/44: Entirely recovered, no recurrence to 12/22/46
4	Chronic "sinus" headache; nasal obstruction	Male, 43	6/11/40 to 9/ 6/47	Postoperative submucous resection	6/11/40	6/11/41: Entirely recovered, no recurrence to 9/6/47
5	"Allergic type"; hay fever and asthma	Female, 41	12/10/42 to 11/20/47	Deviated septum, polypoid degeneration, compensatory changes	1/ 6/43	11/20/47: Free from nasal and "allergic" symptoms
6A	Nasal obstruction in child	Male, 8	1/25/41 to 6/ 5/47	Deviated septum	9/16/41	6/5/47: Free from symptoms, minor septal deformity
6B	Nasal obstruction	Female, 21	1/ 7/43 to 12/28/43	Deviated septum, compensatory changes	1/ 1/43	12/28/43: Free from nasal symptoms
7	Deafness, catarrhal type	Female, 19	10/14/41 to 10/13/44	Deviated septum, spur formation, compensatory changes	10/18/41	10/13/44: Normal hearing (both ears)
8	Deafness, combined type	Male, 43	7/25/41 to 4/ 6/47	Deviated septum, compensatory generalized hypertrophy	7/28/41	4/6/47: 20 decibel recovery; 10 decibel loss remains
9	Deafness, chronic purulent otitis media type	Male, 28	7/30/43 to 8/ 8/45	Deviated septum, compensatory changes	8/ 9/43	8/8/45: Audiogram 15 decibel gain right ear; 15 to 20 decibel gain left ear; 5/17/46; hearing gain maintained

On examination no external deformity of the nose was evident. The septum was deviated to the left, with spur formation along the ridge of the vomer on the left side; this spur made contact with the left inferior turbinate body, which was irregular and large. The left middle turbinate body was crowded and larger than the one on the right. The right inferior and middle turbinate bodies showed considerable compensatory enlargement. A large polypus lay behind the spur in the left middle meatus. There was moderate generalized congestion of the nasal mucosa throughout, with some increase of mucoid secretion, but purulent material was not noted. Transillumination of the frontal and maxillary sinuses revealed them to be fairly clear and evenly illuminated. The tonsils had been

removed. There was considerable granulation tissue along the lateral folds on both sides of the posterior pharyngeal wall.

Local and general treatment was given until the acute condition had subsided. Operation was performed on June 30, with the area under local anesthesia. By means of a modified submucous resection the nasal spur was removed and the remaining bony and cartilaginous portions of the septum were fractured toward the nasal midline (to the right) and the right inferior turbinate body was fractured sharply throughout its entire length. The right middle turbinate body was "notched" with a Gruenwald punch forceps at the most bulbous anterior inferior portion. The polypus was removed from the left middle meatus with the electrocoagulation snare. A single Simpson splint covered with dental wax was inserted into the left inferior meatus to hold the septum in its new position for the first twenty-four hours.

On follow-up one year after operation, the patient had regained his normal weight and was free from all respiratory symptoms. The rheumatic pains were gone, and the general health was good. When last seen, Feb. 15, 1946, the patient had had no recurrence of symptoms and the tissues were normal in color and general appearance.

CASE 2.—*Chronic sinus headache type; chronic rhinosinusitis; chronic nasal obstruction.*

V. W. M., a woman aged 32, was first seen on Jan. 26, 1944. The chief complaint was headache in the frontal area and in the bridge of the nose, radiating to the top and back of the head. She had had frequent colds, one about every six weeks during the year and three during the preceding winter. Additional complaints were of difficulty in breathing through the nose, excessive nasal discharge, feeling of generalized fatigue, sluggishness and mental dulness. She gave a history of nasal symptoms extending back to childhood; she had always had many colds and a great deal of "sinus" treatment. Examination revealed no external deformity of the nose The septum was deviated to the right, and the deformed septum made contact with the right inferior turbinate body. There was slight simple atrophy of the right middle and inferior turbinate bodies, accompanied with compensatory hypertrophy of the left middle and inferior turbinate bodies, together with generalized congestion of the nasal mucosa and increased mucoid secretion. The tonsils had been removed. On objective examination the ears were normal.

By means of a modified submucous resection, done on May 1 with the area under local anesthesia, a septal spur was removed and the remaining bony and cartilaginous portions of the septum were fractured toward the nasal midline (left), and the left inferior turbinate body was fractured outward throughout its entire length.

On follow-up it was learned that the patient had been free from symptoms of headache at the end of one month and since then she has had no further history of colds. When she was last seen, on March 15, 1947, the septum was in the nasal midline, and the mucous membrane was normal in appearance.

CASE 3.—*Reflex referred pain type; chronic rhinosinusitis with deviated septum with spur formation.*

L. J., a 39 year old woman, was first seen on Jan. 17, 1944 reporting the chief complaint of generalized headache with pain in both frontal areas radiating to the top and back of the head and also down the back of the neck into the shoulders. Further complaints were of frequent colds, difficulty in breathing through the nose, excessive nasal discharge, general fatigue, some loss of weight and a sluggish feeling. These symptoms extended back eight years. The patient

had had a great deal of "sinus" treatment. Her teeth had been examined and found normal. Glasses had been prescribed, but they did not relieve the headache. She had been told that she had hay fever, but she had had no tests for allergy. Although examination revealed no external deformity of the nose, the septum was deviated to the left, with spur formation along the ridge of the vomer on the left side. There were compensatory enlargement of the right middle and inferior turbinate bodies, some atrophy of the left middle turbinate body and moderate generalized congestion of the mucous membrane, with considerable increase of mucoid secretion. Transillumination of the frontal and maxillary sinuses showed them to be fairly clear and evenly illuminated. The tonsils had been removed. There was a moderate amount of postnasal discharge adhering to the posterior pharyngeal wall. Objective examination of the ears showed no abnormality except slight retraction of each tympanic membrane. Audiometric examination revealed about a 10 decibel loss of hearing by air conduction in low registers in the right ear and about a 15 decibel loss in approximately the same frequency in the left ear.

At operation, Jan. 18, 1944, done with the area under local anesthesia, the septal spur was removed by means of a modified submucous resection, the remaining bony and cartilaginous portions of the septum were fractured toward the nasal midline (right) and the right turbinate body was fractured sharply outward throughout its entire length. The patient was free from most symptoms at the end of one month, and there was continued improvement without further treatment until Dec. 22, 1946, when the tissues appeared normal.

CASE 4.—*Systemic absorption and chronic "sinus" headache type; chronic rhinosinusitis with septal absorption.*

W. C., a man aged 43, was first seen on June 11, 1940, complaining of dull heavy headaches on the left side of the face in the region of the antrum. He stated that his eyes felt sensitive; there was generalized fatigue of the eyes and impaired "sinus drainage." The history revealed that the present condition had begun twelve years previously with an attack of influenza. He had nasal treatment four times a week for many months. He had had a double antrotomy in 1930 and an operation on the septum in 1933, after which he was advised that further operations would not help. He gave a history of frequent colds; his head always felt dull, and it had been difficult for him to work. He was sensitive to drafts, winds and air-conditioned rooms; he had lost 25 pounds (11.3 Kg.) in weight, had a poor appetite, slept poorly and complained of general fatigue. There was no external deformity of the nose. The septum was markedly to the left, with spur formation along the ridge of the vomer making contact with the middle and posterior thirds of the left inferior turbinate body. There was moderate compensatory enlargement of the right middle and inferior turbinate bodies and some enlargement of the anterior portion of the left inferior turbinate body. A small amount of the inferior border of the anterior tip of both inferior turbinate bodies had been removed at the time of the antrotomy. The openings into both antrums were patent. Partial submucous resection of the septum had been done and part of the triangular cartilage removed. The tonsils had been removed. The ears were found to be normal on objective examination.

Operation was done on the same day, with the area under local anesthesia. By means of a modified submucous resection a revision of the septal operation was done, with removal of the septal spur (left) and the remaining bony and cartilaginous portions of the septum were fractured toward the nasal midline (right) and the right inferior turbinate body was fractured sharply outward throughout its entire length.

Six weeks later there was pronounced symptomatic improvement, with reduced discharge. The patient was gaining weight. On May 6, 1941, there was complete recovery, and there was no recurrence of symptoms up to Sept. 6, 1947, when the nasal tissues appeared normal.

CASE 5.—*Allergic type with hay fever and asthma; pronounced deviation of the septum with compensatory changes in the turbinate bodies, with polypoid degeneration.*

S. M., a married woman aged 41, was first seen on Dec. 10, 1942. The chief complaint was of a "stuffed-up" nose and a feeling of pressure in the nasal regions and ears. In addition, she complained of sneezing attacks, watery discharge from the nose, loss of the sense of smell and loss of head resonance in the voice. Frequent colds had kept her away from work three to five days each month. She was subject to "hay-fever-like" attacks throughout the year, which were worse in summer. She had also had localized attacks of swelling about the face—i. e., half of the upper lip—or of the tissues about one side of her face or about one or both eyes (angioneurotic edema). She complained that she felt tired all the time, did not sleep well, lacked energy, felt mentally dull and believed that her thinking was impaired. She also had asthmatic-like attacks. She gave a history of trouble with the nose since childhood, frequent colds, "stuffy" and "runny" nose, sneezing and coughing. She had had some sort of intranasal operation in childhood, and polypi were removed two years before the present examination. She had had a number of allergic tests with negative results; local nasal treatments had been given over a period of years, but her condition had gradually grown worse. Examination of the nose revealed a slight deviation of the lower portion of the nasal pyramid to the left of the facial midline. The alae were not well dilated; the vestibules are clear. The septum was deviated markedly to the left, so that it almost completely blocked ventilation on that side. A pronounced spur formation along the vomer made contact with the left inferior turbinate body. The left middle and inferior turbinate bodies were somewhat small and atrophied. The right nasal cavity was very much widened because the concavity of the septum was on this side. The right middle turbinate body was greatly enlarged, extending well beyond the nasal midline to the left and making contact with both the nasal lateral wall and the septum. The right inferior turbinate body was greatly enlarged and extended into the septal cavity well beyond the nasal midline. There were several polypi in the right middle meatus. Transillumination of the frontal and maxillary sinuses showed them to be fairly clear and equally illuminated. The tonsils had been removed. Objective examination of the ears showed a slight thickening of each tympanic membrane. An audiogram taken December 12 showed loss of perception of the higher frequencies, beginning at 512 and reaching a loss of 30 decibels at 8,192.

Operation was done Jan. 6, 1943 with the area under local anesthesia. The polypi were removed by the electrocoagulation snare. The mesial half of the left middle turbinate body was removed and the remaining portion fractured outward. The inferior turbinate body was fractured outward along its posterior two thirds. By means of a modified submucous resection, the septum was separated along the base, the spur removed and the remaining cartilaginous and bony portion fractured toward the nasal midline (right) and held in this position by means of a Simpson splint covered with dental wax. On Dec. 14, 1943, two small polypi were removed from the right middle meatus, the right inferior turbinate body was fractured further outward and the septum was again fractured toward the nasal midline. A audiogram taken on December 15 showed normal hearing in

the left ear and considerable improvement in the right ear, except for the 8,192 frequency. On June 7, 1944, the septum was fractured more closely to the nasal midline (right) and right turbinate body fractured farther outward.

On Nov. 5, 1945 the patient reported that she had been free from symptoms for the past six months, and on May 10, 1946 she was still improving. When she was last seen, on Nov. 20, 1947, the septum was in the nasal midline and the nasal mucosa was almost normal in appearance. There were no polypi. She had recovered the sense of smell and was completely free from all allergic symptoms.

CASE 6 A.—*Narrow and obstructive nasal passages in a child.*

J. H., a boy aged 8, was first seen on Jan. 25, 1941. The chief complaint was of frequent colds and stuffy nose, excess purulent discharge, obstructive nasal breathing "sinus attacks," headache, failure to gain weight, poor appetite and generalized fatigue. The present condition had existed for three years but had become worse during the preceding year. Examination of the nose showed rather pronounced deviation of the septum to the right, with almost complete obstruction to ventilation on this side. There was some compensatory hypertrophy of the left middle and inferior turbinate bodies. Generalized congestion of the nasal mucous membrane was accompanied with increased mucoid secretion. The tonsils were present; there was slight injection of peritonsillar tissues. On objective examination of the ears revealed nothing abnormal.

Operation (manipulation only) was done on Sept. 16, 1941, with the patient under general anesthesia. By means of manipulation only, the septum was fractured (with an Asch forceps), mobilized and placed toward the nasal midline (left) by means of the special elevator. A Simpson splint covered with dental wax was inserted into the right nasal passage and removed in twenty-four hours. Recovery was uneventful.

When seen on follow-up Aug. 25, 1945, the boy had had no further treatment and had been free from symptoms since operation. Examination of nose still showed slight deviation of the septum to the right, freedom from discharge, adequate ventilation and color and condition of the membrane within normal limits. On April 2, 1946, he was free from nasal symptoms and there had been no change in physical findings. When last seen, on June 5, 1947, he had continued free from all symptoms and only the minor septal deformity remained.

CASE 6 B.—*Narrow and obstructive nasal passages in an adult; chronic rhinosinusitis.*

J. K., a woman aged 21, was first seen on Jan. 7, 1943, complaining of frequent colds with stuffy nose, excessive postnasal discharge and attacks of laryngitis and hoarseness which interfered with her occupation as singer. The present condition had existed for four or five years, becoming worse during the preceding year. Examination of the nose revealed moderate deviation of the nasal septum, especially in the posterior inferior third. There was some compensatory hypertrophy of the right inferior turbinate body throughout its entire length; the left inferior turbinate body was somewhat enlarged in its anterior two thirds. There was a great deal of mucoid discharge, especially in the inferior meatus on each side. Transillumination of the frontal and maxillary sinuses showed them to be fairly clear and equally illuminated. The tonsils had been removed. There was a moderate amount of granular tissue in each tonsillar fossa, together with some granulation along lateral folds on either side. Objective examination of the ears revealed no abnormalities. Operation (manipulation) was done on

Jan. 1, 1943, with the area under local anesthesia. By means of manipulation both inferior turbinate bodies were fractured outward and the posterior third of the septum was fractured toward the nasal midline (right). At the same time pharyngeal granulations were removed by electrocoagulation. At follow-up on Dec. 29, 1943, the patient was completely free from previous respiratory symptoms and reported no colds since the operation. The septum was in the nasal midline. There was slight generalized congestion of the nasal mucous membrane.

CASE 7.—*Deafness, catarrhal type; deviated and obstructive septum with hypertrophic rhinitis and bilateral chronic catarrhal otitis media.*

N. S., a woman aged 19, was first seen on Oct. 14, 1941, complaining of impaired hearing in both ears, frequent colds and "sinus" attacks. She had first noticed loss of hearing in 1938 while she was in school, and the condition had been gradually getting worse since then. She had had frequent colds since infancy. The tonsils had been removed at the age of 3 years. Local nasal treatment for "sinus" trouble had been given over a period of years. The patient's mother had for many years had impaired hearing of unknown cause. Examination of the nose revealed a very slight external deformity of the nasal bridge, a result of an old trauma; moderate deflection of the septum to the left, with spur formation along the ridge of the vomer; compensatory enlargement of the right middle and inferior turbinate bodies, and thickening and congestion of the entire nasal mucous membrane. Transillumination of the frontal and maxillary sinuses showed them to be fairly clear and equally illuminated. The tonsils had been removed. There was a small amount of granulation at the base of the left tonsillar fossa. The tympanic membranes were intact but moderately retracted. Audiometric examination showed a 40 decibel loss of hearing for low tones in the left ear and a 20 decibel loss in the right ear.

Operation was done in Oct. 18, 1941, with the area under local anesthesia. By means of a modified submucous resection the nasal spur was removed and the remaining bony and cartilaginous portions of the septum were fractured toward the nasal midline (right); both inferior turbinate bodies were fractured outward.

Audiometric examination on March 19, 1942, five months after the operation, showed improvement in hearing, especially in the left ear with no loss for any tone falling below the 20 decibel level. On Oct. 21, 1942, one year after the operation, hearing in both ears was at approximately normal levels. On Oct. 14, 1944, hearing was normal in both ears. The septum was in the nasal midline, and the nasal mucosa was normal in appearance.

CASE 8.—*Deafness, combined type; chronic rhinosinusitis, deviated septum with spur formation.*

J. F., a man aged 43, was first seen on July 25, 1941. His chief complaint was of impaired hearing in both ears, more pronounced on the left, with buzzing and ringing sounds in the head and a sensation of fulness and pressure in the ears. The condition had been of very gradual onset; it dated back five or six years but it was becoming worse and was beginning to interfere with his occupation as waiter. A moderate postnasal discharge of clear mucoid material was noted. The history revealed no involvement of the ears immediately preceding the present condition or in childhood. The patient had never had an aural discharge and did not remember having earache. He had had two or three "colds" each year, but they cleared spontaneously in a week to ten days without headache or "sinus" complications. The tonsils had been removed five years previously, and the patient had had fewer colds since then. There was no external deformity of the nose. The vestibules were open and well formed. There was some deflection of the septum to the left especially in the middle and posterior third. A sharp spurlike formation in the

region of the posterior third of the septum made contact with the posterior third of the left middle turbinate body. The right naris was free from bony obstruction, and the septum showed slight concavity on this side in the middle and posterior portions. There was some compensatory enlargement of the middle and inferior turbinate bodies into the concavity. The nasal mucosa was moderately congested throughout, with more thickening on the right side than on the left. There was a pronounced increase in mucoid secretion, especially in the inferior meatuses. Transillumination of the frontal and maxillary sinuses revealed slightly diminished but equal illumination. The tonsils had been removed. Objective examination of the ears showed the external canals free from deformity or obstruction. The tympanic membranes were intact, and there was no evidence of recent inflammation. A small calcareous deposit was noted in the posterior inferior quadrant of the left membrane. An audiogram showed a conduction and perception loss of about 30 decibels in each ear.

Operation was done on July 28, 1941, with the area under local anesthesia. A modified submucous resection operation was performed, and the spur and ridge along the vomer on the left were removed, and the remaining bony portion of the septum was fractured toward the nasal midline (right). The right inferior turbinate body was fractured outward. A Simpson splint was inserted in the left meatus.

Follow-up investigation revealed that there had been a gradual improvement in the patient's subjective symptoms, with less tinnitus and less feeling of pressure in the ears. An audiogram taken Oct. 8, 1941 showed an average improvement in hearing of about 10 decibels. The improvement continued until the audiogram showed an average loss of less than 10 decibels. The gain in hearing was maintained, the last audiometric examination, on Jan. 3, 1945 showing substantially the same hearing as the audiogram on Jan. 25, 1943. On April 6, 1947, the appearance of the tissues of the nose showed some generalized congestion with moderate excess mucoid secretion.

CASE 9.—*Deafness, chronic purulent otitis media type; chronic bilateral purulent otitis media; chronic rhinosinusitis; deviated septum with nasal obstruction.*

A. S., a man aged 28, was first seen on July 30, 1943. He complained of impaired hearing in both ears, severer on the right, slight tinnitus in both ears and an intermittent discharge from both ears. He reported frequent colds, difficulty in breathing through the nose and excessive postnasal discharge. Further complaints were of a feeling of dulness and heaviness in the "sinuses" and general fatigue. The otic condition had existed since childhood, when there was a discharge from both ears with pronounced loss of hearing. A double mastoidectomy had been performed at the age of 2 years. Recurring mastoiditis in 1922 had been treated with a double mastoidectomy. From age 16 to 25 his hearing had improved, but during last three or four years it had become worse. Examination of the ears showed well healed postauricular mastoidectomy scars on each side. There were large perforations in both tympanic membranes. There was a very slight purulent discharge from the left ear but no discharge from the right ear. An audiogram showed about a 35 decibel loss of hearing in right ear and a 25 to 30 decibel loss in the left ear. The nasal septum was deviated sharply to the left, with compensatory hypertrophy of the right middle and inferior turbinate bodies. Transillumination of the frontal and maxillary sinuses showed them to be fairly clear and equally illuminated. The tonsils had been removed. There was a moderate amount of granulation tissue in each tonsillar fossa.

Operation was done on Aug. 9, 1943, with the area under local anesthesia. A modified submucous resection was performed, with realinement of the septum to the nasal midline and fracture of the right inferior turbinate body outward.

An audiogram made on Aug. 9, 1945 showed an average gain in hearing of 23 decibels in the right ear and of 15 to 20 decibels in the left ear. The patient was free from colds and other nasal symptoms. On May 17, 1946 the right ear was still discharging, but the gain in hearing was maintained. The tissues of the nose appeared normal.

CONCLUSIONS

The forces of compensatory hypertrophy and simple atrophy are used by nature in an attempt to maintain the normal structure of the nose. Through intranasal surgical procedures, physiologic control of the nose can be accomplished by directing these forces.

When the bilateral symmetry of the nose is disturbed, natural adaptations occur which may not be advantageous to the patient; but when the aerodynamic balance is restored through intranasal operations, the normal physiologic processes can be reestablished, partially controlled and directed over a period of time sufficient for effective clinical recovery.

I have developed a simple corrective surgical technic to be applied in the different types of cases with nasal symptoms of varying degree, with or without compensatory changes in the turbinate bodies and contiguous structures, using a biomechanical approach. The compensatory area of the nose is redesigned in order to provide for "maintenance of organic structure" by forming a modified anatomic basis for the compensatory forces, which will then restore the normal physiologic processes. This technic should be applied with great caution in these critical anatomic areas and used only after careful study and complete understanding of the biomechanical principles involved.

The cases reviewed were selected from those of a diversified group of patients of different ages presenting the usual "sinus" symptoms. They include cases of headache, referred pain, allergy (hay fever and asthma) and three types of impaired hearing. All patients showed continued relief from symptoms after varying lengths of time following a corrective operation, including minor follow-up adjustment in some cases. The physiologic functioning of the nose having been directed within natural limits, no further treatment should be necessary.

Drs. Peyton Rous, The Rockefeller Institute for Medical Research, New York; Francis M. Rackemann, Boston; Victor Carl McCuaig, Duncan McCuaig, and Iago Galdston, New York, rendered assistance in this study.

STUDY ON THE RANGE OF USEFULNESS OF A NEW DUAL FORM INTRANASAL MEDICAMENT

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EXCLUSIVE of systemic drugs, there are literally hundreds of nasal medicaments available for the treatment of infections of the upper respiratory tract. These fall into a number of different classifications, such as (*a*) vasoconstrictors, (*b*) vasodilators, (*c*) silver preparations, (*d*) oils, (*e*) antiseptics, (*f*) topical anesthetics, (*g*) volatile agents, inhalers, (*h*) powders, (*i*) estrogens, (*j*) intranasal vaccines, (*k*) cauterizing agents, (*l*) saline solutions, (*m*) bacterial lysates, (*n*) emulsions, (*o*) inhalants, (*p*) nasal jellies, (*q*) nasal sprays and others. In addition, of course, there are the various antibiotics and newer detergent agents. Of vasoconstrictors alone—drops, sprays, inhalants, and jellies—Kully [1] estimated that there are several hundred on the market. As Fabricant [2] has aptly pointed out, of drugs in the classes listed, "many are useful and worthy of clinical trials; some are of questionable value, while others are detrimental to the nose and sinuses."

In the relief and treatment of rhinitis due to the common cold, allergic rhinitis and sinusitis, the physician is confronted with the problem of choosing the most efficacious nasal medicament—and at the same time one that is free from side effects—from among a large variety of widely advertised drugs. What criteria should he use in his selection?

Freedom from interference with ciliary activity is generally held to be a prime requisite of medicaments intended for intranasal application. Second, the preparation should be slightly acid to approximate the p_H of normal mucous membrane. Third, it should be isotonic. And, lastly, it should be free from irritant qualities and from systemic by-effects. Recently, despite the apparent popularity of sulfonamide preparations for local application, the Council on Pharmacy and Chemistry of the American Medical Association [3] has deleted this type of agent from "New and Nonofficial Remedies" because of sensitization occurring in a high proportion of patients treated.

From the Vanderbilt Clinic, Columbia University–Presbyterian Hospital Medical Center; service of Dr. John D. Kernan.

1. Kully, B. M.: Use and Abuse of Nasal Vasoconstrictor Medications, J. A. M. A. **127**:307 (Feb. 10) 1945.

2. Fabricant, N. D.: Nasal Medication: A Practical Guide, Baltimore, Williams & Wilkins Company, 1942, chap. 8, p. 81.

3. Sulfonamides for Local Application Deleted from N. N. R., report of the Council on Pharmacy and Chemistry, J. A. M. A. **135**:157 (Sept. 20) 1947.

It is worth while to emphasize the importance of the proper selection of a vasoconstrictor when one is used, for Lake [4] has recently reported on a condition called "rhinitis medicamentosa," arising from the abuse of nasal vasoconstrictors. He observed that "if the patient has overtreated the nose for only a short time, the turbinates appear only moderately congested. If the patient has overtreated his nose for a long time, the turbinates are congested and the mucous membrane of the nose appears pale, is boggy in texture and has the appearance characteristic of allergic rhinitis." The prevalence of this condition is emphasized by his statement that at the Mayo Clinic, "scarcely a day goes by but that some of us in the Section of Otolaryngology and Rhinology will see one or two patients who are the victims of abuse of nose drops." Many other clinicians [5] have also reported on the increasing incidence of this condition, in which the deeper blood vessels (rather than the subepithelial vasculature) are usually more involved.

That the manufacturers of some of the stronger vasoconstrictors appreciate the dangers attendant on overuse of their preparations on the part of the laity is evidenced by the fact that only recently one of the largest producers withdrew the most potent dosage form from retail drugstore channels and is retaining it for administration by the medical profession exclusively.

According to Lake, treatment of rhinitis medicamentosa consists in discontinuance of local therapy, sedation if necessary and measures designed to "alleviate the original trouble which led to the use of the nasal vasoconstrictors."

CLINICAL EXPERIENCE

In the search for the most efficacious and safe nasal medicament, I have tested a new local preparation [6] in 54 unselected cases of acute and

4. Lake, C. F.: Proc. Staff Meet., Mayo Clin. **21**:367, 1946.

5. (*a*) Clay, J. V. F.: Hahneman. Monthly **81**:60, 1946. (*b*) Gill, J. P.: Eye, Ear, Nose & Throat Monthly **25**:396, 1946. (*c*) Lake. [4] (*d*) Ryan, R. E.: Ann. Otol., Rhin. & Laryng. **56**:46, 1947.

6. The preparation used was narakon,® a product of Baybank Pharmaceuticals, Inc., a division of Chesebrough Mfg. Co. Cons'd. The compositions of the two dosage forms are stated by the manufacturer to be as follows:

Solution Plain	Percentage	Solution with Desoxyephedrine	Percentage
Allantoin	0.40	Allantoin	0.40
Synthetic camphor	0.05	Synthetic camphor	0.05
Synthetic menthol	0.04	Synthetic menthol	0.04
Natural oil of cassia	0.02	Natural oil of cassia	0.02
Sodium monophosphate	1.80	Sodium monophosphatt	1.10
Sodium biphosphate	0.74	Sodium biphosphate	0.50
Benzalkonium chloride 1:3,500	0.23	Benzalkonium chloride 1:3,500	0.22
Distilled water	96.72	Distilled water	96.67
Total	100.0	Total	100.80

chronic conditions of the upper respiratory tract. Patients were taken from my private practice and from the hospital clinic. The test solution was supplied in two forms: plain and with vasoconstrictor. It is a slightly acid, isotonic, aromatized, buffered solution, containing benzalkonium chloride (1:3,500) and allantoin, for use when an antiseptic, detergent, palliative solution (without immediate vasoconstrictive but with gradual decongestive action) is indicated. It is also supplied with added racemic desoxyephedrine (1 per cent) when potent but rebound-free shrinkage is desired.

Controlled bacteriologic tests [7] have demonstrated that both forms of this solution exert marked antiseptic and bactericidal properties against four common organisms, even when diluted. (It is not suggested hereby that the preparation be diluted before administration; these laboratory tests were used merely to check bactericidal efficiency.) With use of isotonic sodium chloride solution for dilutions and testing against the organisms commonly found in the upper airways at 37 C. results were as follows:

Organism	Highest Dilution Killing in 15 Seconds	
	Solution Plain	Solution with Desoxyephedrine
Staphylococcus aureus	1:1	1:1
Corynebacterium diphtheriae	Undiluted	Undiluted
Diplococcus pneumoniae	1:2	1:2
Hemolytic streptococcus	1:2	1:2

Of the 54 patients in the series treated, 28 had sinusitis of various types; 7, rhinitis due to coryza; 5, nasopharyngitis; 2, acute infection of the upper respiratory tract; 5, allergic rhinitis; 3, allergic sinusitis; 1, crusting, dryness and perforated nasal septum; 1, chronic ethmoiditis with polyps, and 2, rhinitis medicamentosa (1 accompanying chronic ethmoiditis and nasal polyps). Twenty-eight patients were given the test solution exclusively; 26 received other medication also.

Of the 28 patients relieved completely by the test solution only, 15 had received diagnoses of sinusitis (6 acute, 9 chronic); 5, rhinitis (2 acute, 2 chronic and 1 with eustachian tube catarrh); 3, nasopharyngitis (2 acute and 1 subacute); 1, acute infection of the upper respiratory tract; 1, allergic rhinitis; 1, allergic sinusitis; 1, dryness, crusting and perforated nasal septum, and 1, rhinitis medicamentosa.

Thirteen of the patients with sinusitis (10 with acute and 3 with chronic) received inhalations of penicillin aerosol in addition to the test solution; 1 with atrophic rhinitis received inhalations of penicillin and vitamin A in addition to the test solution; 1 with acute rhinitis received inhalations of penicillin also—as did 2 with nasopharyngitis, 1 with

7. These tests were made by A. F. Guiteras in the bacteriologic laboratories of Foster D. Snell, Inc., New York.

Clinical Results in 54 Unselected Cases of Acute and Chronic Conditions of the Upper Respiratory Tract Treated with New Test Solution (in Dual Form)

Case No.	Age	Symptoms	Examination	Diagnosis	Dosage,* Drops	Duration of Treatment, Days	Results and Comment
1	34	Nasal congestion, inflamed mucous membrane of nose and throat	Transillumination; antrums and frontal sinuses dark	Acute rhinitis	5 × 3	10	Immediate relief of nasal congestion after 3 days; symptoms free in 10 days
2	23	Stuffy nose; sore throat; swelling of nasal mucosa; postnasal drip	.....	Acute nasopharyngitis	3 × 3	10	Good; nasal decongestion persisted for 2 to 6 hr.; slight "burning" sensation when solution first instilled but marked improvement soon apparent; symptom free after 10 days
3	39	Blocked nose; impaired hearing with noise in right ear	.....	Acute nasopharyngitis and catarrh of right middle ear	5 × 3	22	Nasal decongestion effected; continuous noise in right ear unaffected
4	21	Nasal obstruction; mucous discharge; postnasal drip	Transillumination; frontal and maxillary sinuses clear	Subacute nasopharyngitis	3 × 3	4	Good; nasal condition cleared rapidly after 2 days' treatment
5	16	Headaches above left eye for 2 wk.; purulent nasal discharge	Roentgen; all sinuses clear except left frontal	Acute left frontal sinusitis	5 × 3	8	Subsidence of headache and nasal discharge
6	29	Profuse purulent nasal discharge for a few days, acute swelling of mucous membrane; headache above left eye; deflected nasal septum	Roentgen; some thickening of left antral mucous membrane	Acute left pansinusitis	5 × 3	14	Disappearance of headache and nasal discharge
7	34	Frontal headaches every 3-4 mo. for last 10 yr.; postnasal drip; polyp in left middle meatus	Roentgen; frontal and left ethmoid sinusitis	Chronic sinusitis; pharyngolaryngitis	3 × 3	14	Headaches and nasal discharge disappeared; drops "burned" slightly on instillation; applied with patient prone, letting drops run aganist orifice of frontal sinus; polyps removed
8	40	Pains over and between eyes; mucous membrane of left inferior turbinate engorged	Roentgen; minimal thickening of lining of left maxillary sinus	Chronic rhinitis	5 × 3	21	Headaches relieved; nasal passages temporarily opened
9	55	Congested nasal passages; frequent sore throats; postnasal drip; severe cough	Roentgen; moderate clouding of both frontal sinuses; opacity of both antrums; moderate density of right anterior ethmoid cells	Bilateral chronic frontal and maxillary sinusitis; right ethmoid sinusitis; deflected nasal septum	3 × 3	7	Coughing stopped after 7 days; congestion relieved
10	41	Difficulty in breathing; purulent discharge from nose for 4 days	Transillumination; frontals clear; antrums dark	Bilateral maxillary sinusitis, acute	4 × 3	5	Drops caused slight "burning" but kept passages clear so that patient could sleep; no difference in decongestion observed when another powerful vasoconstrictor was used; 4 penicillin aerosol inhalations of 25,000 units each
11	52	Headaches; nasal stuffiness and discharge	Transillumination; frontals clear; maxillary sinuses dark	Chronic ethmoiditis; nasopharyngitis; incomplete right ethmoidectomy	5 × 3	28†	Good; discharge disappeared; headaches disappeared; 1 penicillin aerosol inhalation of 20,000 units
12	22	"Stuffy" nose	Transillumination; frontals clear; antrums hazy	Acute maxillary sinusitis	4 × 3	5	Good; patient pleased with effect of drops; some discharge still present; 1 penicillin aerosol inhalation of 20,000 units
13	39	"Stuffy" nose	.....	Acute upper respiratory infection	5 × 3	3	Good; improvement in nasal symptoms; 1 penicillin aerosol inhalation of 20,0000 units
14	50	Blocked nose; small nasal polyps	.....	Chronic ethmoiditis with small polyps	4	5	Good; marked relief of nasal symptoms; 2 penicillin aerosol inhalations of 20,000 units each

Clinical Results in 54 Unselected Cases of Acute and Chronic Conditions of the Upper Respiratory Tract Treated with New Test Solution (in Dual Form)—Continued

Case No.	Age	Symptoms	Examination	Diagnosis	Dosage,* Drops	Duration of Treatment, Days	Results and Comment
15	23	Headache; postnasal drip; purulent discharge; deflected nasal septum	Transillumination; frontals clear; right antrum dark	Subacute bilateral sinusitis	3 × 3	25	No discharge visible after 27 days; 7 penicillin aerosol inhalations of 20,000 units each
16	42	"Stuffiness" in nose; postnasal drip; purulent discharge	Transillumination; frontals clear; right maxillary sinus dark	Chronic maxillary sinusitis with acute exacerbation	5 × 3	14	Improvement in nasal breathing and lessening of acute symptoms after 6 days; decongestion of nose and symptomatic improvements; 5 penicillin aerosol inhalations
17	36	Purulent nasal discharge; difficult breathing	Transillumination; frontals and antrums clear	Acute rhinitis; deflected septum	5 × 3	10	Nose completely dry; 2 penicillin aerosol inhalations of 20,000 units each
18	57	Purulent discharge mainly from left nostril; headache	Transillumination; frontals clear; right antrum clear; left antrum dark	Left subacute maxillary sinusitis; myalgia	4-5 × 3	5	Lessening of discharge; headaches subsided; feels considerably better; 2 penicillin aerosol inhalations of 20,000 units each
19	25	"Stuffiness" of nose 8 wk. duration; congested mucous membrane	Transillumination; maxillary sinuses hazy	Chronic nasopharyngitis	5 × 3	2	Excellent; complete relief from all symptoms in 2 days; 1 penicillin aerosol inhalation of 20,000 units
20	14	Purulent discharge from both nostrils after head cold 4 wk. previously	Transillumination; antrums dark	Subacute bilateral purulent maxillary sinusitis	5 × 3	12	Lessening of discharge; 7 penicillin aerosol inhalations and 2 Proetz treatments
21	39	Obstruction of nasal passage; pus in both nostrils for 4 days	Transillumination; antrums and frontals clear	Acute nasal cold	5 × 3	5	Acute condition improved; 2 penicillin aerosol inhalations of 20,000 units each
22	26	Pains in forehead	Transillumination; left frontal sinus dark	Acute left frontal sinusitis	5 × 3 into left nostril	7	Pain and discharge disappeared; 2 penicillin aerosol inhalations of 20,000 units each
23	21	Pain below left eye for 1 wk.	Transillumination; left maxillary sinus dark	Acute left maxillary and ethmoid sinusitis	4 × 3	3	Cleared up; 1 penicillin aerosol inhalation of 20,000 units
24	31	Headaches for 12 yr.; nasal discharge	Transillumination; maxillary sinuses dark	Bilateral chronic sinusitis; deflection of septum to right	4-5 × 3	6	Lessening of discharge and headaches after 6 days; 2 penicillin aerosol inhalations of 20,000 units each
25	48	Occasional headache; purulent nasal discharge for 2 wk.	Transillumination; left frontal and both antrums dark	Acute bilateral maxillary and left frontal sinusitis	5 × 3	4	Cleared up completely; 3 penicillin aerosol inhalations of 20,000 units each and 1 insufflation of sulfadiazine powder
26	39	Offensive-smelling mucus; mucous membrane of both inferior turbinates engorged; allergic	Transillumination; frontals and maxillary sinuses clear	Allergic rhinitis	5 × 3	19	Relief of headaches and nasal congestion
27	30	Attacks of sneezing; watery nasal discharge	Roentgen; frontals show no evidence of disease—clouding of lower right ethmoid cells, thickened lining membranes in both antrums	Bilateral antrum and right ethmoid sinusitis (allergic basis)	3 × 1	7	Nose open; feels well; no complaints
28	42	Clogged nose; pus in both nostrils; pain below right eye	.....	Allergic rhinitis; acute pansinusitis	5 × 3	4	Cleared up in 6 days; prefers test solution to others; 4 penicillin aerosol inhalations of 20,000 units each

29	26	Frequent headaches; postnasal drip; crusting of mucous membranes	Transillumination; antrums and frontals clear	Allergic rhinitis	3 × 3	6	Improvement of postnasal drip; drops were used after penicillin aerosol inhalation treatments, and after test solution was discontinued, anti-histamine tablets were administered
30	86	Chronic nasal stuffiness	Transillumination; frontals clear; antrums hazy	Chronic sinusitis; myocarditis	Tampon soaked with plain solution for 5 min.	11‡	Good; topical application maintained patent nasal airway
31	61	Stuffed nose; mucous membrane covered with pus	Roentgen; heart of hypertensive configuration; sinusitis involving antrums and ethmoid sinuses; sphenoid cells clear; left ventricle enlarged; arteriosclerotic changes in region of aortic knob	Chronic maxillary and ethmoid sinusitis; hypertension	5 (plain) × 3	74	Very good; after 1 week improvement in breathing and decrease in purulent discharge; after 21 days pus dried up and covered mucous membrane in form of crusts; chronic condition greatly improved; no more headaches; after using drops for 74 days felt considerably improved; no side reactions
32	56	Obstruction of nasal airway; purulent discharge	Transillumination; frontals clear; antrums dark	Chronic bilateral maxillary sinusitis; hypertension	10 cc. (plain) on tampons × 1 or 2	42	After trying almost every kind of available nose drops, patient states that test solution relieved clogging of nose and permitted undisturbed sleep at night
33	57	Mucous discharge	Transillumination; sinuses clear	Catarrh of left eustachian tube; acute nasal cold	3 (plain) × 3	5	Mucous discharge of nose subsided
34	51	Dryness and crusting of nose	.....	Perforation of nasal septum	Nasal spray (plain) × 3	3	Dryness and crusting improved
35	55	Complete obstruction of nasal passages through engorgement of mucous membrane; deflected septum	Transillumination; frontals clear; both maxillary sinuses hazy	Rhinitis medicamentosa	Well soaked cotton pledgets in both nostrils (plain) 10 to 15 min. × 2	10	Patient used vasoconstrictors with little effect for 6 months continuously day and night; great improvement in relatively short time with test solution plain and tamponage
36	49	Obstruction of nasal passage	Transillumination; right frontal clear; left frontal cloudy; antrums dark	Acute sinusitis	Tampons (plain) for 5 min. × 2	5	One penicillin aerosol inhalation of 20,000 units; had attack of coronary thrombosis 6 mo. previously; left antrum operated on at age 17; improvement in nasal breathing
37	68	Nasal obstruction and discharge; coughing spells for 2 wk.	Transillumination; frontals clear; antrums dark	Acute sinusitis	Tampons (plain) for 10 min. × 2	4	Nasal passages open; discharge subsided
38	20	Purulent discharge; crusting	Transillumination; frontals clear; maxillary sinuses clear	Deflection of nasal septum; atrophic rhinitis	Nasal spray (plain)	21+	Lessening of crusting; discharge still present; test solution used for over 3 wk.; 5 penicillin aerosol inhalations and vitamin A
39	27	Two day headache; watery nasal discharge; bouts of sneezing	Transillumination; right maxillary hazy; frontals clear	Allergic rhinitis; hypertrophy of posterior ends of both inferior turbinates	Cotton pledgets (plain) 3 to 5 min. (warmed) × 2	7	Helped to keep airway open; allegedly, patient could breathe even while tamponades were in her nostrils; antihistamine tablets and electrocauterization of interior turbinates
40	32	Nose clogged; mucous discharge, occasionally bad smelling, for about 10 yr.; dull headache; frequent colds	Transillumination; frontals clear, maxillary sinuses dark	Chronic sinusitis	5 × 3; changed to plain solution after 6 days	6	Airways open; pus still present in nose, therefore changed to solution plain; 3 penicillin aerosol inhalations of 20,000 units each
41	47	Nasal obstruction; nasal discharge	Transillumination; antrums and frontal sinuses cloudy	Chronic frontal and maxillary sinusitis	2 or 3 × 3	150*	Nasal cavity remained open; drops well tolerated; during this time patient did not use any other intranasal medication

Clinical Results in 54 Unselected Cases of Acute and Chronic Conditions of the Upper Respiratory Tract Treated with New Test Solution (in Dual Form)—Continued

Case No.	Age	Symptoms	Examination	Diagnosis	Dosage.* Drops	Duration of Treatment, Days	Results and Comment
42	26	Clogged nose for several mo.; nasal discharge	Transillumination; frontals clear; maxillary sinuses hazy	Chronic maxillary sinusitis	5 (plain) × 2	27	Nasal discharge stopped, but a slight postnasal drip remained; patient smokes 1 pack of cigarets a day
43	57	Obstructed nasal breathing	Transillumination; frontals clear; maxillary sinuses hazy	Allergic rhinitis	3 to 5 × 1	7	Relief obtained in combination with other treatment; received dust injections and cauterization of turbinates
44	42	Pain above left eye	Transillumination; left frontal and left maxillary sinuses dark	Chronic left frontal and maxillary sinusitis	5 × 3	7	Still some discharge of pus from sinuses
45	45	Discharge from right nostril	Transillumination; frontals clear, right maxillary sinuses dark	Acute maxillary sinusitis; exudate in right middle ear	3 × 3 in right nostril 7 (plain) × 3	1	Relief reported the next day
46	57	Attacks of pain above left eye	Transillumination; left frontal and maxillary sinus dark	Acute left frontal and maxillary sinusitis		7	Blood pressure 190/100; received "empirin"; vasoconstrictor contraindicated but solution plain well tolerated with no side effects
47	39	Nasal discharge; nasal obstruction	Transillumination; maxillary sinuses cloudy	Allergic sinusitis	3 × 2	15	Dust injections and "benadryl" plus test solution relieved allergic complaints
48	21	Obstruction of nasal breathing	Transillumination; maxillary sinuses hazy	Epilepsy; allergic sinusitis; deflection of nasal septum to left	5 × 1	15	Dust injections plus test solution reduced congestion temporarily
49	55	Nasal discharge	Transillumination; frontals clear; maxillary sinuses dark	Chronic maxillary sinusitis	3 × 3	7	Patient has auricular fibrillation, arteriosclerosis and myocardial damage; vitamins administered; nose appeared dry
50	55	Obstructed nasal breathing	.....	Nasal polypo; chronic ethmoiditis; overuse of vasoconstrictors	3 × 3	7	Dust injections and "calcibronat" tablets administered; test solution well tolerated despite rhinitis medicamentosa
51	21	Deafness and noise right ear	Transillumination; sinuses clear	Exudate right middle ear; acute head cold	5 × 3; in right nostril	5	Feels well; symptoms disappeared
52	81	Occasional discharge from nose; formation of crusts	.....	Chronic rhinitis	5 (plain) × 1 in each nostril	10	Crusts and discharge subsided; complains of slight dizziness and headaches 5 min. after instillation of drops; albumin and trace of sugar in the urine and few red cells; blood pressure 175/90
53	62	Deafness and pain in left ear	.....	Acute nasal cold; acute otitis left ear	3 × 3 in each nostril	6	Eardrum looks almost normal; symptoms disappeared
54	47	Nasal discharge; pain radiating into right eye and right molar teeth	.....	Acute right maxillary sinusitis	5 × 2	7	Relief of pain; lessening of discharge

* Unless otherwise specified the solution with desoxyephedrine was used.
† Treatment was intermittent.
‡ Five treatments.

chronic ethmoiditis, 1 with acute infection of the upper respiratory tract and 2 with allergic rhinitis. Antihistamine tablets were administered to 1 patient with allergic rhinitis, and the inferior turbinate bodies were cauterized; another patient with allergic rhinitis received dust injections and was cauterized also; 2 with allergic sinusitis were given dust injections, 1 receiving diphenhydramine hydrochloride (benadryl hydrochloride®) as well, and finally, 1 with rhinitis medicamentosa received dust injections and calcium bromidogalactogluconate (calcibronat® tablets). Full details as to diagnosis, symptomatology, treatment and results are supplied in the table. It will be noted that 39 patients received the test solution with vasoconstrictor; 15, the test solution plain, and 1, the test solution with vasoconstrictor followed by test solution plain. The test solution plain was administered to 6 patients with hypertension, 2 with cardiac involvements and 2 with rhinitis medicamentosa.

RESULTS OF TREATMENT

In my experience, the use of both forms of this solution gave prompt and good results, with complete freedom from side effects in all but 3 cases. Two patients complained of a slight "burning" sensation with use of test solution with vasoconstrictor, but continued its use with benefit; 1 patient, aged 81, with hypertension reported a sensation of slight dizziness and headache five minutes after instillation of drops of test solution plain. It is noteworthy that the 5 hypertensive patients and the 2 with cardiac disease, using the test solution plain, experienced no systemic by-effects.

With both forms of test solution, effective but safe decongestion was achieved; relief from pain and headache was considerable; mucous discharge was lessened; postnasal drip decreased, and dryness and incrustations were markedly ameliorated.

Two cases are of particular interest.

One patient, 61 years of age, used the solution plain continuously (5 drops three times daily) for seventy-four days, with one week's interruption. She suffered from chronic maxillary and ethmoid sinusitis and arteriosclerotic hypertension. Continuous improvement of breathing and reduction in discharge, postnasal drip and headaches were observed. No undesirable local or systemic by-effects were experienced.

Another patient 55 years of age, had had allergic rhinitis for many years, and when she came for treatment her nasal passages were completely obstructed from abuse of vasoconstrictors, which she had used for six months continually, morning and night. Tampons well soaked with the plain solution applied for ten days kept the airways completely open and satisfactorily cleared the superimposed rhinitis medicamentosa.

The second is particularly interesting in view of the general consensus that the most effective treatment for the relief of this condition, as Ryan [5d] stated, is discontinuance of the use of nose drops.

As to form of administration, there appears to be no particular technic which produces superior results. Drops, spray and saturated tampons were used with equal success. In tamponage, the patient was advised to pack only loosely to avert any possibility of ciliary damage. In use of drops, a most important factor is the position of the head; it should be tilted slightly backward and laterally, toward the nostril into which drops are being instilled.

In the 13 patients with sinusitis who received penicillin additionally, the test solution proved helpful in opening the nasal passages for more effective distribution of the penicillin and in preventing the congestion of the mucous membranes often caused by this antibiotic agent.

SUMMARY

A new slightly acid, isotonic, aromatized, buffered, aqueous solution containing benzalkonium chloride (1:3,500) and allantoin, both plain and with desoxyephedrine (1 per cent), was used in the treatment of 54 unselected patients with acute and chronic infections of the upper respiratory tract, including sinusitis, nasopharyngitis, rhinitis and others. Twenty-eight patients received the test solution exclusively; 26 also received other medication.

The solution with desoxyephedrine was administered to 39 patients; the solution plain was administered to 15 and both forms to 1. It was used in the form of drops, spray and saturated tampons.

Duration of treatment varied from one day (in a patient with acute maxillary sinusitis) to over seventy days (in a patient with chronic maxillary ethmoid sinusitis with arteriosclerotic hypertension).

Results were good in most cases, with pronounced relief of pain, headache and nasal "stuffiness"; mucous discharge was lessened; postnasal drip decreased, and dryness and incrustations improved greatly. In the 13 patients with sinusitis who received penicillin additionally, the test solution proved helpful in opening the nasal passages for more effective distribution of the penicillin and in preventing the congestion of the mucous membrane frequently caused by this antibiotic agent.

There was no evidence of irritation of the mucous membrane or any indication of allergic sensitization, except in 3 of the 54 patients. Two patients complained of a slight "burning" sensation with the use of the test solution with vasoconstrictor, but continued its use with benefit. There were also no systemic reactions, such as cardiac disturbances, nervousness or insomnia, nor other evidence of toxicity (except in 1 hypertensive patient aged 81, who complained of slight dizziness five minutes after instillation of the test solution plain). It is especially

noteworthy that the solution plain proved completely safe for the 5 patients with hypertension (1 patient used it for seventy-four days), 2 with cardiac disease and 2 with rhinitis medicamentosa.

From my experience, it appears that the test solution in the plain form should prove particularly helpful in those cases in which ephedrine and ephedrine-like substances are contraindicated, as in diseases of the upper respiratory tract complicated by hypertension, arteriosclerosis, vasomotor rhinitis, pregnancy, old age, etc. It may also be safely and beneficially used over long periods in treatment of sinusitis and other chronic conditions for its antiseptic, detergent, apparently mildly astringent and emollient properties. The form containing desoxyephedrine is valuable in cases in which potent, rebound-free antiturgescent medication is indicated. After the vasconstrictor form has provided ventilation and drainage in the acute phase, any needed subsequent treatment may be continued with the solution in plain form.

Administration of the test solution in either form does not, of course, preclude the need in some cases for systemic or other medication aimed at relieving or eliminating the underlying cause.

TYMPANOSYMPATHETIC ANESTHESIA FOR TINNITUS AURIUM AND SECONDARY OTALGIA

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TINNITUS aurium is frequently a symptom of disease of the inner ear and often a symptom of disease of the middle ear. In the absence of an active pathologic process in the ear, tinnitus is the result of a constitutional disorder. While the exact physiologic mechanism of this distressing symptom has not been explained satisfactorily, a number of theories have been proposed, none of which, however, has been proved therapeutically.

In the appraisal of all aural symptoms, tinnitus is the most distressing, and it often is of such annoyance as to cause the patient constant torture. This distressing situation produces an inability to concentrate and prevents the carrying out of routine daily duties. In addition, it often becomes worse during the night and prevents adequate sleep and rest. In many persons tinnitus finally produces mental depression and other mental disorders.

Tinnitus aurium per se is a symptom localized to the ear and should not be confused with head noises in general. True tinnitus aurium, or intrinsic tinnitus, is subjective and remains localized to the ear.

It differs from head noises, or extrinsic tinnitus, in that head noises move about in the head, so that the patient may complain of noises in the forehead, in the top of the head, in the ears and at the base of the skull.

A careful history as to the nature and characteristics of the noise is important in the differential diagnosis of tinnitus. Fowler [1] classified tinnitus as vibratory, caused by actual autogenous vibrations reaching the ear from any part of the body, and nonvibratory, caused by biochemical irritation of the auditory neural mechanism. It is the nonvibratory, or intrinsic, type with which this study is concerned. While nonvibratory, or intrinsic, tinnitus may gradually become louder after

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1. Fowler, E. P.: Head Noises and Deafness: Peripheral and Central, Laryngoscope **49**:1011 (Oct.) 1939; Tinnitus Aurium in the Light of Recent Research, Ann. Otol., Rhin. & Laryng. **50**:139 (March) 1941.

weeks or months, wide daily variations are exceptional. This is not true of extrinsic tinnitus, or head noises, which varies greatly from day to day and often undergoes frequent variations in character and intensity on certain days. The subjective noises of intrinsic tinnitus take the form of high-pitched tones—chirping, singing sounds, the sound of escaping steam or boiling water, ringing, buzzing, pulsating sounds and the long shrill notes of the cicada.

Since intrinsic tinnitus is obviously a symptom of neural involvement occurring in disorders of the middle and the internal ear, it is logical to assume that the neural elements of these structures must play a part in the production of this annoying symptom. Fowler [2] also

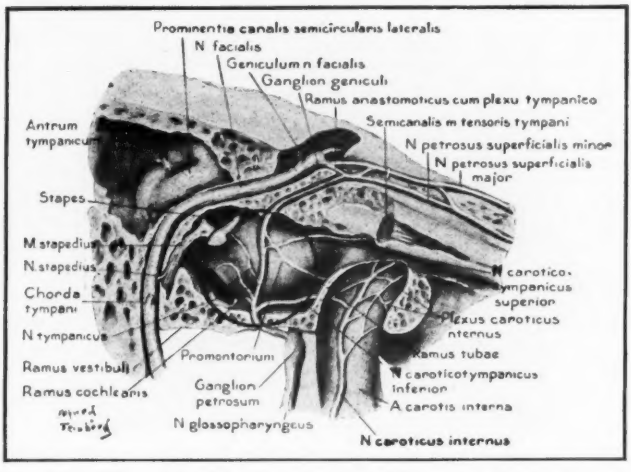

Fig. 1.—The tympanic plexus and its rami vestibuli and cochlearis (after Lempert [3]).

stated that nonvibratory (intrinsic) tinnitus is caused by irritation of the auditory nerve or, more properly, the auditory neural elements. Furthermore, it has been observed that there is an intimate nerve connection between the neural elements of the middle ear and those of the internal ear. The tympanic plexus, which constitutes the neural elements of the middle ear, is connected with the cochlear neuromechanism of the internal ear through the ramus vestibuli and the ramus cochlearis (fig. 1).

2. Fowler, E. P.: Nonvibratory Tinnitus, Arch. Otolaryng. **47**:29 (Jan.) 1948.

Lempert [3] described the tympanic plexus as arising from the sensory fibers of the trigeminus, sympathetic and glossopharyngeal nerves (fig. 2).

Jacobson's nerve, which arises from the glossopharyngeal nerve, is the most highly developed in the tympanic plexus. This nerve contains isolated ganglion cells. It passes from the fossa jugularis through an opening in the inferior tympanic wall and, extending upward in a groove in the promontory, anastomoses with the small superficial petrosal nerve, which arises from the trigeminus.

The sympathetic nerves of the lining membrane of the middle ear arise from the sympathetic plexus accompanying the carotid artery. Several small branches of this plexus enter the tympanic cavity through orifices in the carotid canal as the superior and inferior caroticotym-

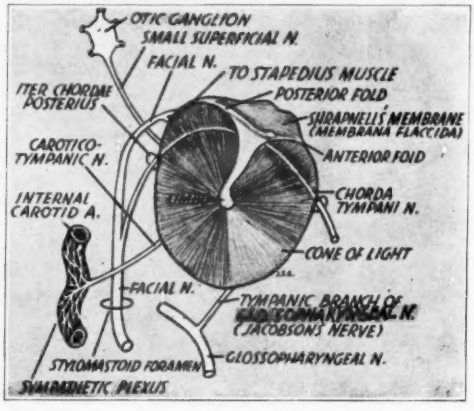

Fig. 2.—Origin of the tympanic plexus from the fifth nerve (small superficial petrosal), the ninth nerve (Jacobson's) and the carotid sympathetic nerves. After Barnhill.

panic nerves. These, in conjunction with the small branches of Jacobson's nerve and the small superficial petrosal nerve arising from the trigeminus, form the tympanic plexus in the anterior portion of the tympanic cavity. Arising from this plexus are smaller nerve branches, which supply the entire lining membrane of the middle ear and in which one finds, in the regions of the fenestra vestibuli and fenestra cochlearis, clusters of ganglion cells. In addition, a delicate, ramifying network of nerve fibers is found, which spreads partly above and partly below the vessels and forms ganglionic swellings where several fibers meet.

3. Lempert, J.: Tympanosympathectomy: A Surgical Technic for the Relief of Tinnitus Aurium, Arch. Otolaryng. **43**:199 (March) 1946.

A large branch arising from the tympanic plexus runs also to the cartilaginous eustachian tube.

Through this connection of the trigeminus, sympathetic and glossopharyngeal nerves, which form the tympanic plexus, tinnitus aurium may be produced by pathologic changes in the middle ear and in neighboring structures, such as the teeth, pharynx, nose and eustachian tube. It seems to make little difference whether the active lesion is located in the middle ear or in the neighboring structure, since the impulse which produces tinnitus is transmitted or relayed from the middle ear to the inner ear by the tympanic plexus. Apparently, the tympanic plexus acts as a central nerve plexus, receiving impulses through its connections with the ninth nerve, the fifth nerve and the carotid sympathetic fibers. These impulses are manifested either by tinnitus or by aural pain. Presumably, the intimate connections of the tympanic plexus with the inner ear through the ramus vestibuli and the ramus cochlearis create two phases in aural response to stimulation. Impulses of a minimal nature traverse the tympanic plexus without initiating a reaction of pain but are adequate to stimulate a reaction of tinnitus in the sensitive cochlear neuromechanism. Finally, if the stimulation becomes more intense, a reaction of pain occurs through stimulation of the tympanic plexus itself. The initial phase of minimal irritation is a subthreshold phase, resulting in tinnitus, and the secondary phase is a suprathreshold phase, resulting in pain.

While histologic investigations so far have not definitely demonstrated and established the neural connections between the tympanosympathetic fibers and the cochlear neuromechanism, there is much clinical and experimental evidence to support the belief that they exist. Observations made in the course of suppurative inflammations of the middle ear, and pharyngeal, lingual and dental infections suggest a definite physiologic relation between tinnitus aurium and secondary or referred otalgia. The following clinical observations tend to confirm the presence of these neural connections and their importance in producing tinnitus aurium and reflex otalgia from involvement of the middle ear and its neighboring structures.

The association of tinnitus with acute suppurative otitis media, particularly in its early stages, is commonly observed. Furthermore, it is observed that otalgia and tinnitus aurium frequently coexist in the presence of acute inflammatory lesions of the middle ear. When inflammation of the middle ear subsides, tinnitus and otalgia subside; this occurrence leads to the conclusion that the physiologic structures within the middle ear involved in otalgia and in tinnitus aurium are one and the same, or are intimately related in some manner. In cases of acute suppurative inflammation of the middle ear spontaneous or induced drainage immediately diminishes otalgia and tinnitus. The mechanism by which tinnitus and otalgia are produced can be compared with that of

any inflammatory process. For example, drainage of an abscess alleviates pain because pressure within the abscess cavity is reduced. Complete relief from pain is obtained when the inflammatory reaction within the surrounding tissues has disappeared.

The same process of inflammatory edema and pressure on neural fibers produces tinnitus and otalgia in the middle ear cavity. Since the tympanum is more or less in direct connection with the cochlea through the fenestra vestibuli, and especially through the fenestra cochlearis, the effect of fluid pressure from the middle ear on these fenestrae must be evaluated in the production of tinnitus. It is common belief that the accumulation of inflammatory exudates in the middle ear and the effect of their pressure on the fenestrae initiate tinnitus in the cochlear mechanism. In order to observe the effect of fluid pressure within the middle ear, experiments were performed with patients who were entirely free of tinnitus. Isotonic sodium chloride solution was injected into the middle ear through the intact tympanic membrane with a fine caliber hypodermic needle so that the tympanum was completely filled with fluid. The effect of pressure of the fluid was accentuated by having the subject incline the head to the side so that the fluid covered the fenestrae to the internal ear. In only 1 patient was tinnitus or otalgia induced by fluid pressure in the middle ear. It is evident, therefore, that tinnitus and otalgia are not caused by pressure effects alone but are the result of the inflammation of some anatomic structure within all or a part of the tympanum. Apparently, inflammatory involvement of the tympanic plexus is responsible for these symptoms. In suppurative otitis media inflammation of the mucosa of the promontory involves the tympanic plexus in an inflammatory process, which, in turn, produces tinnitus and otalgia. Purulent exudation in the middle ear further increases tinnitus and pain by the effect of pressure on the inflamed tympanic plexus. Draining of the middle ear by myringotomy or spontaneous rupture partially alleviates pain and tinnitus through removing pressure on the involved tympanic plexus. Further reduction in tinnitus or otalgia occurs as the inflammatory edema involving the tympanic plexus resolves with the disappearance of the infection. In view of these findings, and of Lempert's observations on the otoneurology of the tympanic plexus, in which he noted clusters of ganglion cells in the regions of the fenestra vestibuli and the fenestra cochlearis, there apparently is a definite relation between the histopathologic state of the tympanic plexus and the symptom complex of tinnitus. When ganglionitis occurs in the region either of the round or of the oval window, a tonus impulse is transmitted to the inner ear which is interpreted as tinnitus. These clinical observations when correlated with the onset and recession of tinnitus indicate that the tympanic plexus is the initial source of the stimulation leading to tinnitus from inflammation of the middle ear.

Clinical observations made on numerous other pathologic conditions further substantiate the belief that the tympanic plexus is a definite segment in the neural pathway which produces tinnitus. The anatomic connections and reflex pathways that are associated with referred otalgia are well known. While tinnitus is an equally common symptom in such conditions, little effort has been made to explain its neural pathways. In view of the identical pathologic conditions producing tinnitus and otalgia and the similarity of onset and recession of these symptoms, it is suggested that the same neural pathways may be responsible for them. The well known pathologic conditions that produce otalgia or tinnitus, or both, are acute suppurative sialadenitis, chronic sphenoiditis, chronic adhesive otitis media, acute occlusion of

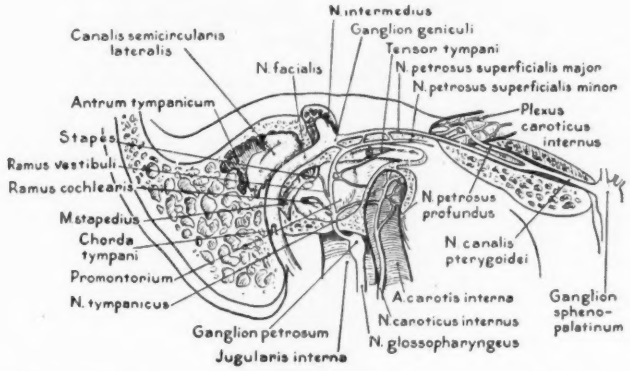

Fig. 3.—Neural pathway of tinnitus and otalgia from the nose and sinuses via the sphenopalatine ganglion. After Lempert.

the eustachian tube, abscessed teeth, ulcerative lesions of the pharynx or of the larynx and inflammation of the nose and sinuses.

Figure 3 illustrates the neural pathway of secondary or referred otalgia and tinnitus arising from chronic sphenoiditis and other inflammations of the nose and sinuses. Impulses are transmitted from the sphenopalatine ganglion through the greater superficial petrosal nerve to the tympanic plexus to incite tinnitus via the connecting ramus vestibuli and ramus cochlearis or to produce aural pain in the tympanic plexus itself.

The course of reflex pain or tinnitus from an abscessed tooth is by way of the inferior dental nerve through the auriculotemporal nerve to the otic ganglion and then to the tympanic plexus through the lesser superficial petrosal nerve (fig. 4).

Acute occlusion of the eustachian tube and ulcerative lesions of the pharynx and of the posterior third of the tongue giving rise to tinnitus or otalgia do so by reflex pathways through the glossopharyngeal nerve (fig. 5).

One particularly common condition producing tinnitus or aural pain through the glossopharyngeal nerve is ulceration of the faucial pharynx following tonsillectomy.

Circulatory disturbances of the internal carotid artery are responsible for tinnitus in a large number of persons. The pulsating, or throb-

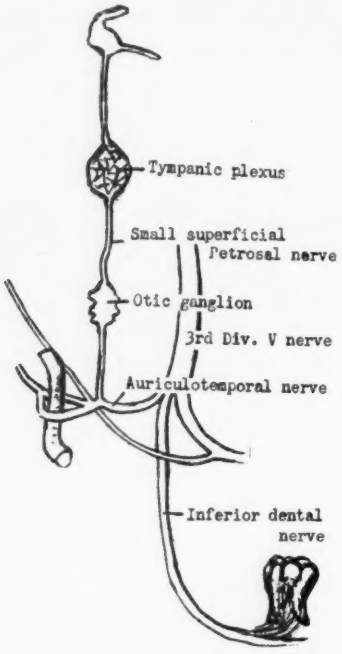

Fig. 4.—Neural pathway of tinnitus and otalgia from an abscessed tooth. After Barnhill.

bing, type of tinnitus is characteristic of these vascular disturbances. The acoustic effect of vascular changes in the carotid artery can be easily demonstrated by digital pressure on the carotid plexus in the neck. Tinnitus can be initiated or, if present, changed in character by digital pressure on the carotid plexus.[2] The reflex pathway is through the plexus caroticus to the plexus caroticus internus, and then by way of the nervus caroticus tympanicus inferior and nervus caroticus tym-

panicus superior to the tympanic plexus, where the impulse is transmitted to the cochlea through the ramus vestibuli and the ramus cochlearis (figs. 1 and 3).

Familiar examples of intracranial vascular disturbances which set up or accentuate tinnitus through the sympathetic caroticotympanic mechanism are straining at stool; interference with cranial circulation, such as occurs in the head-down position, constriction at the waist, such as occurs during bending action, and constriction at the throat, such

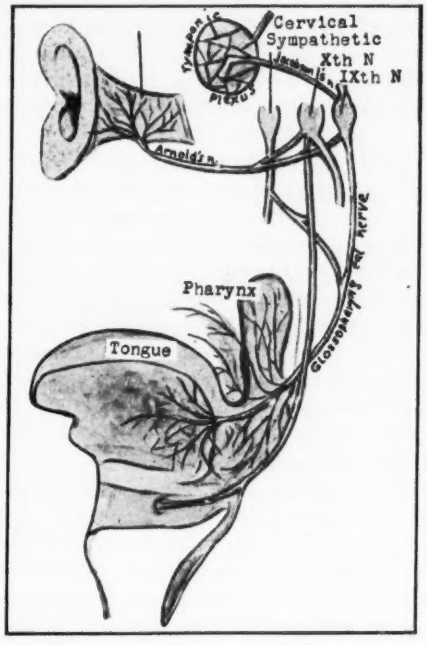

Fig. 5.—Neural pathway of tinnitus and otalgia from the tongue and the pharynx. After Barnhill.

as occurs with wearing tight neck bands. One of the more constant causes or accompaniments of tinnitus is arteriosclerosis.

According to Lempert, when ganglionitis involves the sympathetic caroticotympanic filaments coming from the carotid plexus, a tonus impulse is transmitted to the inner ear, where it is interpreted by the organ of Corti as pulsating tinnitus.

Another likely source of vascular tinnitus is the middle meningeal artery. In figure 4 it is observed that two roots of the auriculotemporal nerve form a complete loop around the middle meningeal artery. This

peculiar neural arrangement occurs just below the foramen spinosum, through which the middle meningeal artery enters the middle fossa of the cranial cavity. The inaccessibility of this region to neurosurgical approach and to postmortem study has resulted in little information being gained concerning the relation of the middle meningeal artery to the symptom complex of tinnitus aurium. The anatomic arrangement of the auriculotemporal nerve in this location is similar to that of the left recurrent laryngeal nerve as it loops around the arch of the aorta. Laryngeal conditions resulting from vascular abnormalities of the aorta as they affect the left recurrent laryngeal nerve are well known. Presumably, in a similar manner, vascular abnormalities of the middle meningeal artery produce tinnitus through the auriculotemporal nerve. The neural impulses are transmitted by the auriculotemporal nerve to the otic ganglion and then to the tympanic plexus through the lesser superficial petrosal nerve.

Another particularly significant observation on the role of the tympanic plexus in relation to tinnitus is the "peach glow" discoloration (Schwartze sign) of the promontory seen in otosclerosis. Often the initial symptom of otosclerosis is an annoying and distracting tinnitus. In many instances, especially early in the disease, there is observed through the tympanic membrane a "peach glow" or "flamingo red" tinge, indicative of hyperemia in the cochlear promotory, or of thickening and increased vascularity of the mucosa of this region, in which the tympanic plexus is located. These observations strongly suggest a relation between the histopathology of the tympanic plexus and the symptom complex of tinnitus.

While the histologic and anatomic structure of the tympanic plexus, with its distribution of branches, filaments and ganglionic swellings, lends itself to a reasonable explanation of intrinsic tinnitus, it must be remembered that there exist sound observations on tinnitus with relation to the histopathology of the eighth nerve and lesions of higher nerve centers. These various observations on the explanation of tinnitus in no way discredit one another but give rise to an original concept that intrinsic tinnitus may originate either centrally or peripherally with the cochlea as the focal neuromechanism of response. Common sources of central intrinsic tinnitus are acoustic neurofibromas, lesions of the cerebellopontile angle and some drug allergies.

The histopathology of drug sensitivity is not clear in its relation to the production of tinnitus. Heretofore, the effect of quinine, salicylates and the arsenicals in the production of tinnitus was thought to be of central origin. However, observations on the effect of quinine [4]

4. Bishop, S. S.: Diseases of the Ear, Nose, and Throat and Their Accessory Cavities, ed. 3, Philadelphia, F. A. Davis Co., 1905, p. 522.

showed that it produced congestion of the middle ear, as well as of the labyrinth. This observation indicated that tinnitus could arise through congestion of the middle ear, involving the tympanic plexus; hence, tinnitus of this nature would be of the peripheral intrinsic type.

On the basis of some of the aforementioned histologic, anatomic and clinical observations, experiments in injection of the tympanum to eliminate tympanosympathetic tinnitus have been carried on during the last seven years. A preliminary report [5] on the use of ethylmorphine hydrochloride for this purpose was published some years ago. Since then, several revisions in technic have greatly improved the results of tympanic injection. While in pharmacologic classification, ethylmorphine hydrochloride U. S. P. is an analgesic, it also exhibits anesthetic characteristics when injected into the tympanum. This drug has proved to be effective in the treatment of tinnitus and secondary otalgia because of its combined anesthetic and analgesic qualities.

The therapeutic value of ethylmorphine hydrochloride in removing pupillary exudates and in absorbing interstitial corneal deposits by producing pronounced inflammatory edema has long been known in ophthalmology. It acts as a vasodilator and lymphagogue, stimulating the vascular and lymphatic circulation of the eye and producing pronounced dilatation of the blood vessels and lymphatics. It is for these physiologic effects on the tissues of the promontory and the tympanic plexus that this drug is used in injections into the tympanum. Ethylmorphine hydrochloride is particularly effective for this purpose because it has a selective action on neural tissue. Immediately after injection of the drug into the tympanum, the inflammatory reaction the solution produces can readily be observed through the otoscope. The entire drum membrane, particularly Shrapnell's membrane, becomes congested. Although definite inflammatory discoloration of the drum membrane occurs, the landmarks are not obscured by edema. The fluid line of the ethylmorphine hydrochloride solution can easily be seen through the tympanic membrane. The patient experiences little discomfort, since the drug produces analgesia, which counteracts the discomfort of the injection and the subsequent inflammation. This factor is of practical importance, as the patient is not incapacitated and is able to continue at his daily occupation, without discomfort. The therapeutic effect of the drug injected into the tympanum results from its analgesic action on the tympanic plexus and from its rehabilitating action on the tissue of the middle ear, by stimulation of vascular circulation and the subsequent absorption of interstitial fibrosis.

5. Trowbridge, B. C.: Injection of the Tympanum for Chronic Conductive Deafness and Associated Tinnitus Aurium: A Preliminary Report on the Use of Ethylmorphine Hydrochloride, Arch. Otolaryng. **39**:523 (June) 1944.

SELECTION OF CASES

If good results are to be obtained, the cases for injection must be properly selected. The following points should be noted:

1. The tinnitus must be of the peripheral intrinsic type that has been described.

2. Pronounced degeneration of the acoustic nerve with associated long-standing tinnitus is only occasionally benefited by this type of therapy.

3. Peripheral intrinsic tinnitus associated either with apparently normal hearing or with defective hearing are benefited by this therapy.

4. Peripheral intrinsic tinnitus of one year's duration or less responds best to tympanosympathetic anesthesia.

5. Peripheral intrinsic tinnitus resulting from acute otitis media has not been treated by this method while in the acute stage. However, good results have been obtained during the subsiding stage or in cases of subacute otitis media. Injections are started two weeks after the acute stage.

6. The tympanic membrane may or may not be intact. Peripheral intrinsic tinnitus with or without a history of previous inflammation of the middle ear is amenable to this therapy.

7. Unilateral peripheral intrinsic tinnitus responds best to injection therapy. Bilateral intrinsic tinnitus is usually of central origin and hence does not respond to tympanosympathetic anesthesia.

8. Intractable otalgia from degenerative or ulcerative lesions of the eustachian tubes, nasopharynx, faucial pharynx, tongue, jaws, teeth or nose is benefited by injection of the tympanum.

9. The external auditory canal must be normal, without cellulitis, furunculosis, eczema or other disorder.

10. The vestibular mechanism must be normal as tested by the caloric method.

11. There is no definite age limit. The injection of 4 minims (0.25 cc.) of a 5 per cent solution of ethylmorphine hydrochloride is equivalent to 1/6 grain (10 mg.) of the drug.

MATERIALS AND METHODS

Tympanosympathetic anethesia is accomplished by injecting 4 minims (0.25 cc) of a 5 per cent solution of ethylmorphine hydrochloride into the tympanum through the posterior inferior quadrant of the tympanic membrane.

Materials and instruments which are necessary for tympanic injection are a cotton wick, for topical anethesia of the drum membrane, a 1 ounce (30 cc.) bottle of an aniline oil–cocaine solution; a rubber-capped ½ ounce (15 cc.) bottle of a sterile 5 per cent solution of ethylmorphine hydrochloride; a 1 cc. tuberculin syringe with a no. 22 spinal (Quincke point) 3 inch (7.6 cm.) needle and a magnifying otoscope, or a magnifying lens insert, in a head mirror (fig. 6).

Prior to injection into the middle ear cavity, anesthesia of the ear drum membrane is produced by local application to the tympanic membrane of an aniline oil–cocaine solution (chemically pure aniline oil, 90 parts; cocaine hydrochloride U.S.P., 10 parts). A cotton wick saturated in the aniline oil–cocaine solution is applied to the posterior inferior quadrant of the drum membrane for ten minutes; after removal of the wick, the external auditory canal is wiped dry. In order to avoid contamination of the middle ear cavity subsequent to injection, the use of free anesthetic solution in the aural canal is not recommended.

The patient lies on a treatment table with the ear to be injected directed upward. The head is slightly rotated laterally, so that the eustachian tube of the ear to be treated is directed upward. This is done to prevent the escape of the solution from the middle ear through the eustachian tube, into the throat. With a 1 cc. tuberculin syringe and a no. 22 spinal (Quincke point) 3 inch needle, 4 minims of a 5 per cent solution of ethylmorphine hydrochloride is injected into

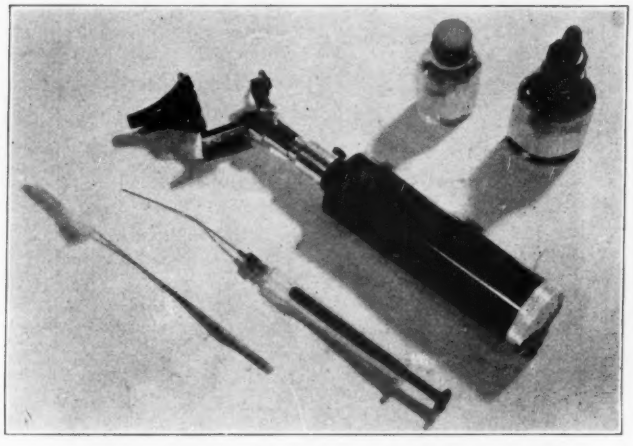

Fig. 6.—Materials and instruments for tympanic injection.

the tympanum through the tympanic membrane just below and posterior to the umbo (fig. 7).

The patient maintains his position for five minutes after the injection. Occasionally, transient vertigo follows the injection. Injection of the tympanum by this method is surprisingly free from uncomfortable or disturbing reactions. After the instillation of the solution, relative analgesia occurs, which lasts for hours. Patients seldom complain of any painful sensation but, rather, note of a feeling of fulness in the ear. Inflammatory changes in the tympanic membrane can be observed within a few minutes after the injection. All inflammatory signs subside within four days after the injection.

The treatment consists of successive instillations of a 5 per cent solution of the drug at intervals of four days. Peripheral intrinsic tinnitus and referred otalgia may be completely, and apparently permanently, abolished in the majority of cases by the injection of 4 minims of the solution on five consecutive four day intervals. No definite rule can be made governing the number of injections to be

made, since patients differ in their response to them. Some patients require fewer than five tympanic injections; others, who show only slight improvement after a series of five injections, should be allowed a rest for two months, after which a second series of five injections is given. Some patients have dramatic results, with complete loss of tinnitus or otalgia, while in others, with partial benefit, relief from these annoying symptoms is sufficient to permit normal activity and sleep.

Before beginning injection therapy for tinnitus, an audiometric analysis should be made. A careful audiometric examination is essential to determine the type

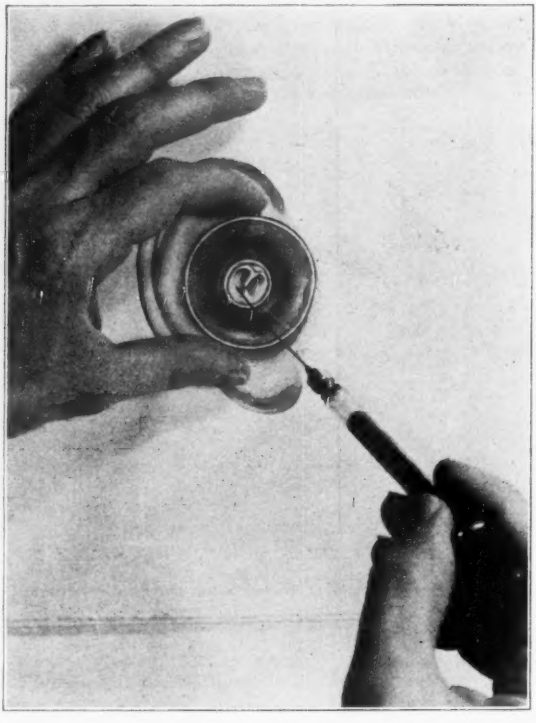

Fig. 7.—Tympanic injection.

and the amount of hearing defect, if present, as well as to aid in the identification of the type of tinnitus.[6] Follow-up audiograms of patients who had injection therapy have shown definite improvements in their hearing.[5] Apparently, there is nothing more effective in decreasing tinnitus than in diminishing deafness. Tympanic injection therapy accomplishes both.

6. Trowbridge, B. C.: Electroaudiography: Analysis and Interpretation of the Audiogram, Arch. Otolaryng. **35**:899 (June) 1942.

RESULTS

Of a series of 20 cases selected for injection of the tympanum to alleviate tinnitus or otalgia, good results were obtained in the majority (table).

In 9 of the cases listed in the table there was not complete loss or cessation of tinnitus or otalgia. However, in the majority of cases

Results of Tympanic Injection in Treatment of Tinnitus Aurium and Secondary Otalgia

Case	Age, Yr.	Type of Tinnitus and Pain	Duration	Etiologic Factors	Condition of Drum Head	No. of Injections	Results
1	55	Roaring, right	2 wk.	Spontaneous onset	Mild catarrh	5	Complete relief
2	44	Ringing, bilateral	4 yr.	Gradual onset	Mild catarrh	2	Slight improvement
3	54	Buzzing, bilateral (left, worse)	6 mo.	Spontaneous onset	Mild hyperplasia	5	Slight improvement
4	40	Buzzing, left	8 yr.	Gradual onset	Hyperplasia	4	Pronounced improvement
5	59	Roaring and intermittent pain, right	12 days	Myringitis bullosa hemorrhagica, right	Lusterless	4	Complete relief, hearing improved
6	61	Cricket sound, right	15 yr.	Gradual onset	Mild hyperplasia	5	Tinnitus less severe at night
7	44	Whistling and intermittent pain, right	6 wk.	Acute rhinitis	Retraction	3	Complete relief
8	30	Buzzing, right	6 mo.	Spontaneous onset	Mild catarrh	1	Complete relief
9	36	Pain, left	1 wk.	Abscessed left lower first molar	Mild catarrh	1	Complete relief
10	47	Peanut stand whistle, left	1 wk.	Acute rhinitis	Hyperplasia	1	Complete relief
11	33	Ringing, with loss of hearing, left	2 yr.	Gradual onset	Hyperplasia	3	Pronounced improvement
12	34	Shooting pain, left	4 days	Post-tonsillectomy	Mild catarrh	1	Complete relief
13	41	Cracking, right	4 yr.	Frequent infection of upper respiratory tract	Hyperplasia	2	Moderate improvement
14	63	Roaring, with loss of hearing, left	2 wk.	Acute recurrent maxillary sinusitis	Adhesive otitis media perforation, healed	3	Complete relief, hearing improved
15	24	Intermittent shooting pain, left	4 days	Extraction of left lower tooth root	Mild catarrh	1	Complete relief
16	19	High pitch tinnitus, left	1 yr.	Traumatic rupture of ear drum membrane	Perforation, healed	1	Complete relief
17	68	Sizzling, and steady earache, right	3 mo.	Acute rhinitis	Hyperplasia	1	Complete relief, hearing improved
18	25	Buzzing, left	4 yr.	Recurrent infection of upper respiratory tract	Adhesive otitis media	3	Moderate improvement
19	65	Singing and intermittent pain, left	25 yr.	Gradual onset after tonsillitis	Hyperplasia	3	Tinnitus diminished, pain ceased
20	49	Ringing, bilateral	2 mo.	Gradual onset	Hyperplasia	6	Moderate improvement, left

the tinnitus was decreased to the point where it was no longer a constant source of annoyance. (Most of the patients without complete relief stated that the tinnitus had diminished sufficiently that it did not interfere with their sleep or mental activities.) In 2 cases no improvement was obtained. In a previously reported series of 22 cases [5] in which the effect of injection therapy on hearing acuity was primarily studied,

improvement in hearing occurred in 18. In 4 cases no improvement was obtained. Of 9 of the cases in which there was associated tinnitus aurium, lessening of the tinnitus occurred in 6 and loss of the tinnitus in 3. The better results obtained in the present cases than in the previously reported series with regard to tinnitus are due to improvements in the technic of injection since the original series was reported.

All the patients were ambulatory and continued their usual daily routines without interference from the injections. Since many patients drove automobiles, the effect of tympanic injection on equilibrium had to be considered. Vertigo rarely occurred after the injections. It was not necessary for the few patients who manifested slight transient vertigo to suspend normal activity; they were able to continue their normal activity after several minutes. Sustained vertigo was not observed in any case. Other secondary reactions were negligible. Acute pain and secondary suppuration of the middle ear did not occur after injection in any case. While there is general reluctance to pierce the intact tympanic membrane, not one case of residual perforation occurred. Closure of the site of injection occurred immediately, with formation of a small, petechia-like blood clot marking the point of injection. An interesting observation which was made independently by Dr. Brownell [7] and myself was that of the improved appearance of the tympanic membrane after a series of tympanic injections. Hyperplastic and lusterless drums regained their semitransparency and their pearly sheen.

SUMMARY AND CONCLUSION

Injection of the tympanum offers a new approach to the therapy of tinnitus aurium and referred otalgia. While there is not one medical or surgical method that will cure all types of tinnitus, tympanic injection is useful in alleviating peripheral intrinsic tinnitus, the commonest type of tinnitus. In the majority of cases the intensity of tinnitus can be reduced to a point where it is no longer a source of major irritation. Since this minor surgical procedure is directed toward correcting the effect of aural and related lesions, one must not fail to locate and remove, if possible, the cause of the disturbance.

In case 9 of the table are demonstrated the factors of "cause" and "effect" of reflex impulses through the tympanosympathetic plexus. In this case reflex otalgia was initiated by an abscess of the first left lower molar tooth. Otalgia was completely eliminated by one injection of the tympanum. This procedure is not often necessary for this particular cause of otalgia, since the source can be easily located and removed. However, in case 9 is demonstrated the pathway of reflex impulses to the

7. M. E. Brownell, M.D., senior resident in otolaryngology, University of Kansas School of Medicine, assisted in the clinical studies.

"trigger" area, the tympanosympathetic plexus, in the middle ear. Injections into the tympanum for referred otalgia arising from ulcerative and degenerative lesions, which are less amenable to direct therapy, give much relief from pain. Since peripheral intrinsic tinnitus is evidently a subthreshold phase of secondary or referred otalgia, the same methods of treatment apply to its relief and removal. The favorable results obtained in the treatment of tinnitus and secondary otalgia by tympanic injection suggest the following classification of tinnitus and its secondary phase of otalgia.

Types of Tinnitus
- Extrinsic tinnitus (vibratory)
  - (*a*) Psychoneurotic
  - (*b*) Psychosomatic (menopause)
  - (*c*) True vibratory
- Intrinsic tinnitus (nonvibratory)
  - Peripheral (subthreshold phase)
    - (*a*) Tympanic plexus
    - (*b*) Trigeminal nerve { Otic ganglion / Sphenopalatine ganglion }
    - (*c*) Glossopharyngeal nerve
    - (*d*) Carotid sympathetic nerves
  - Central
    - (*a*) Acoustic nerve to nuclei
    - (*b*) Supranuclear pathways

Secondary otalgia (suprathreshold phase)
- Reflex or referred pain
  - (*a*) Tympanic plexus
  - (*b*) Trigeminal nerve { Otic ganglion / Sphenopalatine ganglion }
  - (*c*) Glossopharyngeal nerve
  - (*d*) Carotid Sympathetic nerves

Mechanism of Tinnitus and Secondary Otalgia

Initial phase (of minimal irritation)—a subthreshold phase resulting in tinnitus.

Secondary phase—a suprathreshold phase resulting in pain

Tympanic injection producing tympanosympathetic anesthesia is a simple and practical procedure for the relief of tinnitus aurium and secondary otalgia in properly selected cases. Aural pain or tinnitus will subside or completely disappear if the trigger zone, the tympanosympathetic plexus, is removed early. By temporarily interrupting the tinnitus or pain reflex pathway, tympanosympathetic anesthesia stops the flow of impulses. No harmful after-effects occur, and side reactions are negligible.

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UVULA AND TONSILS

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THE QUESTION of what role the uvula plays under normal conditions has stimulated the interest of many laryngologists. Yet only a few answers, and these tentative, have been given in the past.

A paper by Richardson and Pullen,[1] recently published in these ARCHIVES, attributed so many physiologic functions to the uvula that it seems to be a very important organ of the human body. However, it remains to be seen whether further observations will prove that the conclusions drawn by Richardson and Pullen from their experience are entirely justified. They concluded that the uvula massages and moistens the posterior pharyngeal wall and aids in removing and moving downward material from this wall, that it transfers the secretion from the midline of the velum and the posterior pharyngeal wall from above and anteriorly to the midline posteriorly, that it is a valuable aid in guarding against middle ear disease in children and that, by preserving the normal pharyngeal arches, it prevents pharyngitis sicca, with subsequent drying of the eustachian orifice. A deviation of the uvula to one side may indicate weakness of the accessory nerve on the other side. According to these authors, the uvula prevents rhinolalia aperta (hyperrhinolalia) as a complication of successful tonsillectomy. In my opinion this statement is tenable only under exceptional conditions, namely, if the soft palate is so short that it can reach the posterior wall of the pharynx only by using its entire length and the uvula. In this case the uvula may partly fill the gap between the pharyngeal raphe and the contracted constrictor fibers. But, since the uvula is either oval or conic while the gap is angular, the uvula will not always fill the space. Furthermore, it has been proved that there may be a maximum distance of 3 mm. between the posterior pharyngeal wall and the soft palate without hyperrhinolalia being present. If the soft palate is not abnormally short, there is, as is well known, a part hanging down even if the soft palate rises to touch the posterior wall of the pharynx. The part hanging down is the perpendicular part, and it is this portion to which the uvula is attached. The perpendicular part, however, neither creates nor prevents hyperrhinolalia.

1. Richardson, G. S., and Pullen, E. M.: The Uvula, Arch. Otolaryng. **47:** 379-394 (April) 1948.

Normal, as well as abnormal, conditions of the uvula may help in determining the physiologic role or roles of that organ. I presume that it is well known to many laryngologists that the uvula shrinks after tonsillectomy. I have devoted several studies to this occurrence and have published the results.[2] My first article was based on an observation on only 75 patients. Since then I have examined hundreds of patients, some of them both before and after the tonsillectomy, and

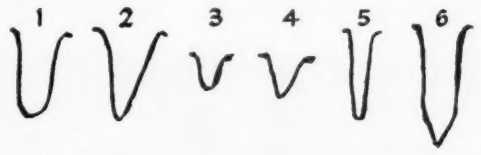

Fig. 1.—Six types of the normal uvula.

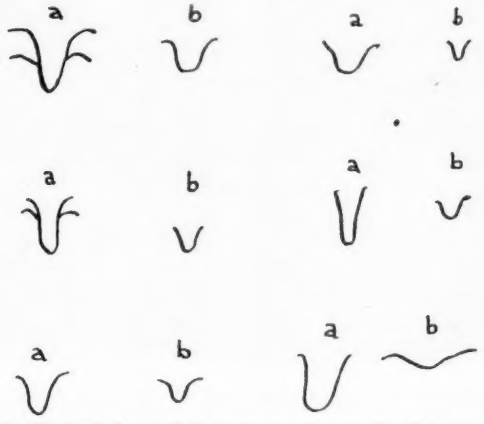

Fig. 2.—Uvulas before and from two to three weeks after operation.

others only after the operation. All these observations have confirmed my former findings.

The uvula under physiologic conditions may present one of six types, as shown schematically in figure 1. Of 278 normal uvulas, 157 belonged to type 1, 46 to type 2, 56 to type 3, 8 to type 4, 8 to type 5 and 3 to type 6. Of 250 persons with tonsils removed, only 14 showed type 1, 8 type 2, 11 type 3 and the rest type 4. In all patients the

2. Froeschels, E.: Ueber das Schicksal des Zäpfchens nach Entfernung der Gaumenmandeln, Monatsschr. f. Ohrenh. **69**:155-158, 1935.

uvula had the normal form of its respective type but was smaller. No fixed relation could be found between the size before and that after the operation. Frequently, a very large uvula shrinks to a very small one, but in rare cases the reduction in size is not great. Yet some shrinkage was noticeable in all of the patients observed before and after tonsillectomy. Even if one has not had an opportunity to observe a patient before tonsillectomy, the uvula afterward is so conspicuously short, and frequently so thin, in the great majority of cases that one is inclined to assume a reduction in size. Sometimes the edges of the

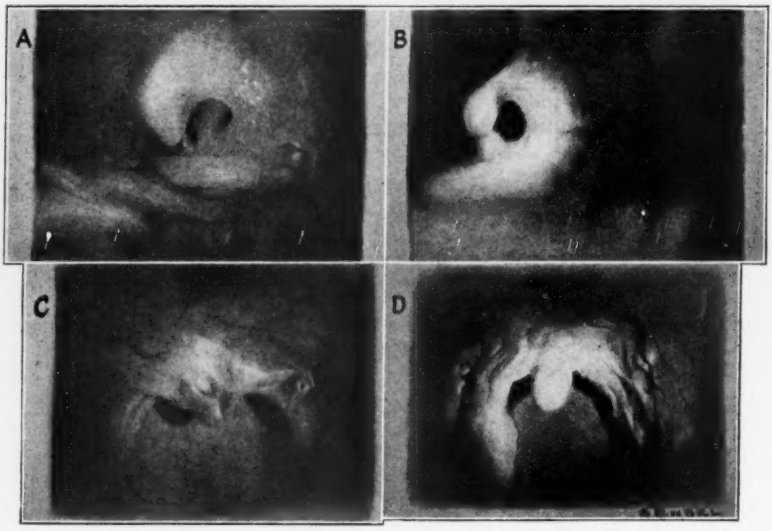

Fig. 3.—*A*, photograph of an uvula before operation; *B*, the same uvula after operation; *C*, uvula of a cadaver with tonsils removed; *D*, arches and uvula of a cadaver with tonsils.

uvula are almost transparent, a condition which to my knowledge does not occur under normal conditions.

In my first publication on this subject I stated that it is hard to determine just how long after the operation the shrinking began because the usual swelling in the region of the faucial arches hinders clear judgment. It was stated in this article that two weeks after the operation the diminution in size was visible in every case. Later, in several cases, I had the opportunity of seeing the uvula three or four days after tonsillectomy. In these cases the edges were discolored and sometimes looked almost transparent. Yet it was impossible to decide whether edema or diminution of the normal tissue or both were

responsible for the peculiar appearance. One case suggested the possibility that the shrinking process may last a long time. A woman reported to me that since her tonsils had been removed, four years before, her uvula had been getting progressively smaller.

Figure 2 shows sketches of 6 uvulas before and from two to three weeks after the operation. *A* and *B* of figure 3 present photographs of a uvula before and after tonsillectomy.[3] *C* and *D* of figure 3 are photographs of the uvula and arches of 2 cadavers. Figure 3 *C* shows a cadaver with tonsils removed; *D*, the uvula of a cadaver with the tonsils not removed. The uvula in figure 3 *C* is so small that the size can be attributed to the shrinking after tonsillectomy, although no comparison with its preoperative size can be offered.

Histologic examination of 4 uvulas from cadavers with tonsils removed showed that no real atrophy was present and that none of the normal tissue was missing or reduced out of proportion as compared with other physiologic tissue of the organ. In other words, histologically, a shrunken uvula looks exactly like a normal uvula of the same size.

It is not certain that decrease in size is due to the diminution in circulation of blood caused by scar formation, for the blood supply derives partly from arterial branches coming from the tonsillar region to the soft palate and partly from the sphenopalatine artery (anterior supply). Furthermore, persons with visible and severe scars in the arches do not show particularly small uvulas.

Not enough is known about the functions of the tonsils. Only recently Šercer and Ruždíc [4] published a paper on the sugar-fermentative function of the tonsils. Perhaps the tonsils are not without influence on the metabolism of neighboring organs.

Two practical conclusions may be drawn from the observations described: first, that tonsillectomized persons offer an excellent opportunity for comparison of the functions of normal uvulas and those of shrunken uvulas; second, that in cases—certainly rare—in which there is an indication for tonsillectomy and for clipping the uvula, the latter may be postponed until the shrinking of the uvula has taken place, thus making the clipping superfluous.

133 East Fifty-Eighth Street (*22*).

3. Unfortunately, one side of the pharynx is invisible, but different sizes of the uvula are well recognizable.

4. Šercer, A., and Ruždíc, J.: Beiträge zur Kenntnis der fermentativen Tätigkeit der Tonsillen, Acta oto-laryng. **36**:236-249, 1948.

Case Reports

PERFORATION OF THE ESOPHAGUS BY A THUMB TACK AND SUBSEQUENT REMOVAL BY AN ELECTROMAGNET

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The following case report is considered of interest because mediastinitis following perforation of the esophagus by a foreign body was treated successfully and because the foreign body was removed by means of an electromagnet.

REPORT OF CASE

The patient, a 10 year old boy, was admitted to the hospital at 6 p. m., April 18, 1947. At 2:45 p. m. that day he had swallowed a thumb tack. His only complaint was that of a sensation of something sticking in his throat. Physical examination was noncontributory.

Lateral and anteroposterior roentgenograms of the neck (fig. 1*A*) revealed the outline of a thumb tack lodged in the anterior wall of the esophagus at the level of the sixth cervical vertebra. The shaft of the tack was directed anteriorly and slightly to the right of the midline.

Soon after admission, with the patient under ether anesthesia, the esophagus was explored with an esophagoscope. As no evidence of the tack could be found after a thorough search of the hypopharynx and the entire length of the esophagus, roentgenograms were taken with a portable machine. The wet films did not reveal the foreign body. Figure 1*B* shows that the tack was discernible on later examination, after the films were processed and dried.

Penicillin therapy (30,000 units of penicillin every three hours) was instituted on the patient's return to his room.

The following morning the child appeared to be ill. The respiratory rate was 20 per minute; the pulse rate, 80, and the rectal temperature, 101 F. Roentgenologic examination at this time disclosed that the tack was in the esophagus and that the head of the tack had become separated from the shaft (fig. 1*C*). The shaft was at the level of the third thoracic vertebra, with the point of the tack directed anteriorly and slightly to the right of the midline. The head of the tack was at the level of the sixth thoracic vertebra. It ultimately passed through the gastrointestinal tract. An area of increased density in the upper part of the middle lobe of the right lung was noted.

The patient was having some dysphagia. By noon the rectal temperature had risen to 102 F, the pulse rate to 96 and the respiratory rate to 24. Respirations were of the grunting type. The leukocyte count was 18,750 per cubic millimeter of blood, with 88 per cent neutrophils, and the concentration of hemoglobin was 13.5 Gm. per hundred cubic centimeters. At this time the child complained that he was having increased difficulty with swallowing and that he had pain in the lower right side of his neck. Physical examination revealed a slight swelling at the base of the neck on the right side in the supraclavicular area. Roentgenograms taken at this time (three hours subsequent to that in fig. 1*C*) revealed that the pulmonary area of density in the middle lobe of the right lung had

increased. An area of widening of the hilus toward the right was also noted. Otorhinolaryngologic and surgical consultants decided on supportive treatment, chemotherapy and use of antibiotics. Administration of penicillin was continued on a three hour schedule with increased dosage (100,000 units for the first two doses and 50,000 units thereafter). Intravenous administration of sodium sulfadiazine was begun (0.5 Gm. every four hours).

For the next two days the child was ill, even irrational at times, and the pain he complained of previously persisted. In the region of pain were swelling, tenderness and induration. Fever was of the septic type, with the rectal temperature rising to 103 F. The pulse rate was 100. The respiratory rate was 26, and respirations continued to be of the grunting type. The leukocyte count decreased to 12,700, with 82 per cent neutrophils. Roentgenologically the position of the shaft of the tack was not changed, but the width of the mediastinum had increased one and a

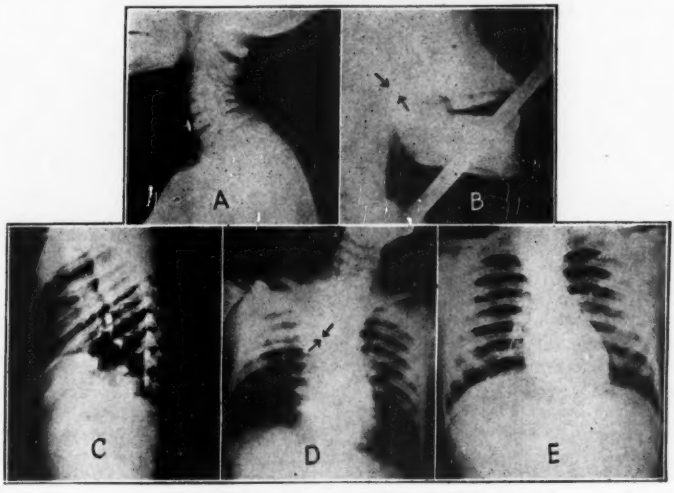

Fig. 1.—*A*, lateral roentgenogram taken on the patient's admission; *B*, roentgenogram at time of esophagoscopy; *C*, roentgenogram taken twelve hours later, on following morning; *D*, anteroposterior view, showing increase in width of mediastinum; *E*, roentgenogram of chest on nineteenth postoperative day.

half times its normal size (fig. 1*D*). A zone of density, corresponding to gas, extended superiorly from the level of the tack shaft.

On the fourth day the patient was more alert, complained less of pain and took large quantities of fluid by mouth without difficulty. However, the swelling at the base of the neck on the right side increased. There was increase in the temperature and in the pulse and respiratory rates. The leukocyte count decreased to 10,000, with 89 per cent neutrophils. The mediastinum was widened further. In the lateral view the major portion of this area of increased density appeared to be located posteriorly, but also extended anteriorly.

In the next two days, his fifth and sixth days in the hospital, clinical improvement continued. The swelling at the base of the neck increased and evidenced

beginning fluctuation. A roentgenogram showed a definite extension of the mass in the right side of the mediastinum. In the lateral view there appeared to be some compression of the lower portion of the trachea.

On the seventh day, with the patient under intratracheal nitrous oxide–oxygen and ether anesthesia, a 4 inch (10 cm.) vertical incision was made parallel to the sternocleidomastoid muscle on the right over the neurovascular bundle. The latter was retracted laterally, the middle thyroid vein and inferior thyroid artery were ligated, the lobe of the thyroid was retracted medially and the dissection was carried medially and inferiorly. An abscess cavity was entered at about the level of the suprasternal notch between the cervical vertabrae and the esophagus.

Fig. 2.—Patient four months after operation. There is a birthmark in the left sternoclavicular area.

Approximately 200 cc. of white, gelatinous, odorless fluid was released from this cavity in the mediastinum. Similar material was, at the same time, aspirated from the mouth by the anesthetist, an indication that there was simultaneous drainage of the abscess through the esophagus.

Two Penrose drains were inserted into the base of the abscess cavity and were anchored at the edge of the wound.

Culture of the aspirated material gave a scanty growth of a hemolytic streptococcus and an abundant growth of nonhemolytic Staphylococcus aureus.

Postoperatively, water taken by mouth streamed through the wound in the neck, with frothy saliva. Intravenous supportive treatment was instituted. On the sixth

postoperative day a Levin tube was passed through the nose to the stomach for feeding, and two days later fluid ceased to drain through the incision in the neck.

Laryngoscopic examination revealed that the right vocal cord was fixed slightly lateral to the midline.

Recovery was uneventful, and the temperature rapidly fell by lysis. A roentgenogram on the sixth postoperative day showed that the mediastinal shadow had definitely decreased. The lateral view revealed the shaft of the tack to be in the mediastinum posterior to the esophagus, as outlined by the Levin tube.

On the ninth postoperative day, with the aid of a fluoroscope, a flexible laryngeal forceps was passed through the wound in the neck to the bottom of the abscess cavity. The shaft of the tack appeared to be only a few millimeters from the tip of the forceps. A large electromagnet was then attached to the forceps, and shortly thereafter the point of the tack appeared to be in contact with the side of the forceps. The forceps was then slowly withdrawn with the shaft of the tack attached.

The wound ceased to drain three days later. A roentgenogram on the nineteenth postoperative day disclosed little residual evidence of the episode (fig. 1*E*).

Examination of the larynx four months later indicated that mobility of the right vocal cord was restored. Swallowing was normal; roentgenographic studies with barium sulfate showed no abnormalities, and the wound in the neck was closed (fig. 2).

COMMENT

The complications in this case—the change in position of the tack, the separation of the head from the shaft and the injury of the recurrent laryngeal nerve—as well as the subjective improvement under treatment with penicillin and sulfadiazine, are of interest.

On the day the patient was admitted to the hospital, the foreign body could not be found in the course of a thorough esophagoscopic examination, nor was it evident in the roentgenograms taken at the time. However, the nasopharynx was not examined. It seems likely, then, that the tack was regurgitated into the nasopharynx during the induction phase of anesthesia and was lodged there during esophagoscopy. In the recovery phase of anesthesia the tack was again swallowed and separated, the shaft penetrating the esophagus and the round head passing on through the gastrointestinal tract. The faint outline of the tack could be seen after the films taken with the portable machine were passed through the dryer (fig. 1*B*).

Sodium sulfadiazine and penicillin produced subjective improvement and decrease of the leukocyte count; however, mediastinitis progressed, as evidenced by fever and abscess formation.

Injury of the recurrent laryngeal nerve during drainage of the mediastinal abscess resulted in transient paralysis of the right vocal cord. At examination of the larynx two months later function of the right vocal cord was found to have returned completely.

248 Poppy Street, Corona Del Mar, Calif.

Clinical Notes; New Instruments and Technics

IMPROVED METHOD OF APPLYING A MEDICINAL SOLUTION TO THE MIDDLE MEATUS OF THE NASAL CHAMBER

BRUNO L. GRIESMAN, M.D.
NEW YORK

THE ADMINISTRATION of nasal medication usually has in view two objectives: first, shrinkage of the congested mucous membrane with the aim of restoring proper nasal breathing; and second, shrinkage of the region of the ostiums of the sinuses with the purpose of establishing drainage. The problem of eradicating infection at its site is not considered in this paper.

"Before nasal medication can attain maturity, a number of perplexing problems require solution" (Fabricant [1]). The question how to deliver a medicinal solution to those areas of the nasal chamber where it will be most effective is frequently asked by the practicing physician, and the problem raises a number of pertinent considerations.

If one looks at the base of the nose without using a nasal speculum, only raising the lobule with a finger, one will notice the columella and the nasal septum in the midline and in the nostril laterally the bulging head of the inferior turbinate; also, the inferior meatus and part of the floor of the nose may be seen. However, in most cases it is impossible to see the middle turbinate or the middle meatus. These anatomic parts of the nasal cavity are hidden by the upper lateral cartilage, the caudal end of which projects into the vestibule as a significant landmark. Several authors call this line by different names: the limen nasi or the plica vestibuli.

Figure 1, a drawing of the lateral nasal wall, shows the limen nasi (*a*). This important landmark runs from a point in the nasal tip just behind the caudal end of the septal cartilage laterally toward the attachment of the inferior turbinate. If one intends to administer a medicinal solution to the middle meatus, it seems evident that the tip of the applicator should be placed behind and above the plica vestibuli.

The inferior meatus plays no important role in nasal medication, since the sinus openings are situated in the middle and superior meatuses. The maxillary sinus, the anterior ethmoid cells and the frontal sinus drain into the middle meatus. Figure 1 shows these sinus ostiums in the lateral nasal wall. The posterior ethmoid cells and the sphenoid sinus communicate with the superior meatus. The ostium of the maxillary sinus is located above the level of the floor of the sinus and cannot drain in the erect position of the head. The ostium of the frontal sinus and that of some grooves of the ethmoid sinus point downward and favor natural drainage. Therefore, in cases of acute and chronic sinusitis it is

From the Department of Otolaryngology of the New York Polyclinic Medical School and Hospital.

1. Fabricant, N. D.: Nasal Medication, Baltimore, Williams & Wilkins Company, 1942.

the middle meatus which should be reached by local nasal medication. However, most applicators used do not serve the purpose.

Figure 1 demonstrates the possible direction of the flow of fluids in the nose. Arrows point to the inferior (*a*) and the middle (*b*) meatus. Fluid administered to the inferior meatus often drains backward along the floor of the nasal cavity into the pharynx. In other cases fluid runs out of the nostril in which the applicator is located, and is thus inefficiently administered. Frequently, in order to help the fluid reach the middle meatus, the patient is required to assume an awkward position and perform irksome gymnastics. Parkinson [2] recommended the head-low posture to enable medication to reach the ostiums of the nasal sinuses.

To overcome these drawbacks, I attempted to construct an applicator which would permit the administration of nasal medication directly to the middle meatus and which could be used easily and comfortably by adults and children.

Figure 2 *A* is an illustration of the new applicator. The letter *a* indicates the body portion of the applicator and *b* the nozzle, or outlet portion, while *c*

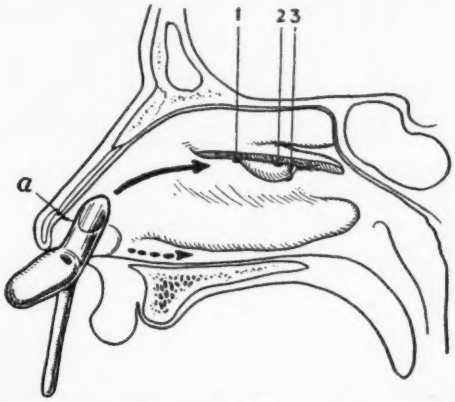

Fig. 1.—Drawing of a sagittal section of the nose. The middle turbinate bone has been removed, exposing the sinus openings: *1*, ostium frontale; *2*, ostium ethmoidale; *3*, ostium maxillare. The drawing also shows the position of the applicator, the opening of which can be seen above the limen nasi (*a*).

indicates the handle by which the applicator is conveniently manipulated. The body portion (*a*) provides a chamber in which the fluid to be administered is contained, the same being delivered into the interior of the body through the open end (*d*), out of which it is also delivered into the middle meatus.

It is to be noted that the upper edge of the nozzle (*b*) is slanted or inclined, thus providing the nozzle with a sidewall (*e*) that is higher than the opposite wall (*f*). The top wall of the body (*a*) is provided with a small hole, or air vent (*g*).

The operation of the new applicator will be readily comprehended from figure 2 *B* and *C*. The number of desired drops is delivered to the body through the

2. Parkinson, S. N.: A Lateral Head-Low Position for Nasal and Sinus Treatment, Arch. Otolaryng. **17**:787 (June) 1933.

opening (*d*) with an ordinary nasal dropper or by being poured through the opening. The applicator is held substantially horizontally in the hand by the stem (*c*), and the nozzle portion (*b*) is inserted into the nostril near the tip of the nose above the plica vestibuli. In inserting the nozzle (*b*) the longer wall portion (*e*) of the nozzle should be directed toward the upper cartilaginous

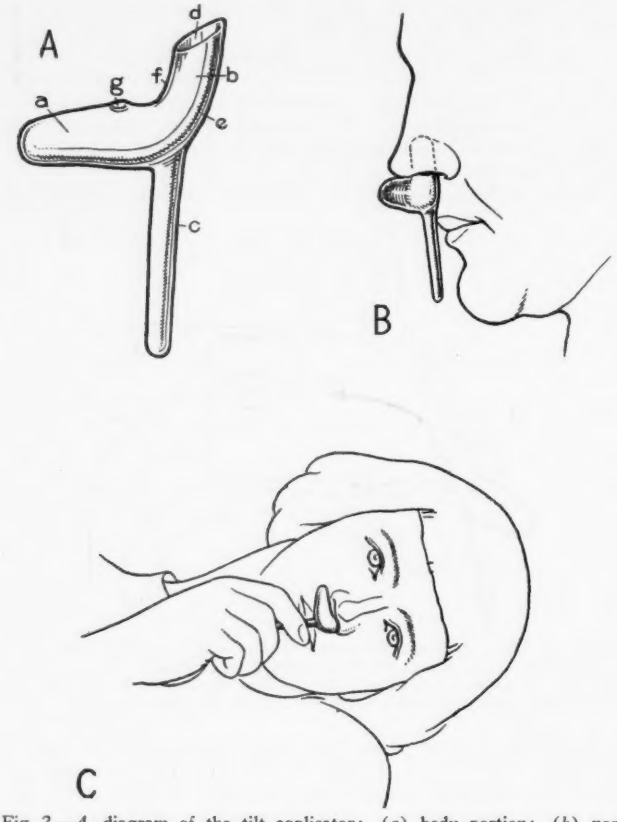

Fig. 2.—*A*, diagram of the tilt applicator: (*a*) body portion; (*b*) nozzle; (*c*) handle; (*d*) open end; (*e*) higher side wall; (*f*) opposite lower side wall; (*g*) air vent.

B, the position of the head with the applicator installed.

C, the tilting of the head toward the shoulder to release the fluid from the applicator into the middle meatus.

vault, i e., the lateral wall of the external nose, and the short wall (*f*) of the nozzle should be located toward the nasal septum.

By placing a finger on the outside of the external nose, one can feel the exact position of the tip of the applicator. Figure 1 shows this position in relation to

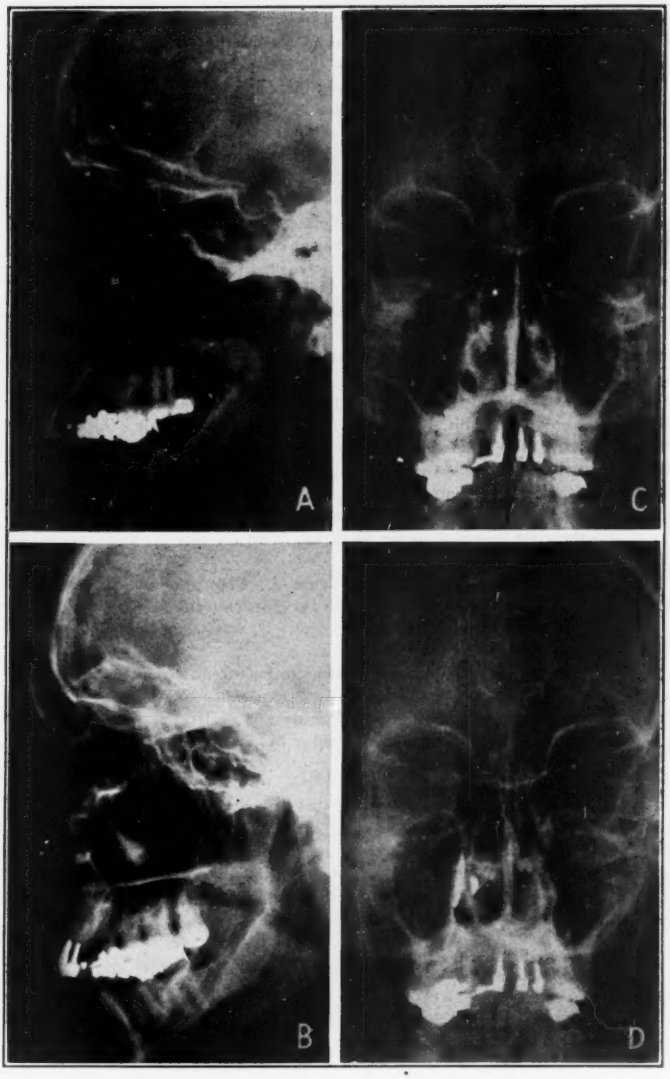

Fig. 3.—Roentgenograms taken before and after instillation of iodized poppy-seed oil 28 per cent: *A* and *C* were taken ten minutes after administration of a vasoconstrictor and before instillation of the iodized oil. *B* and *D* were taken after instillation of the oil. *D* was taken five minutes before *B*.

the lateral nasal wall. The position of the opening of the applicator can be seen above the limen nasi, directed toward the middle meatus. The body portion (*a*) of the applicator extends crosswise below the columella.

Holding the applicator so applied, the patient's head (fig. 2 *B*) is first tilted slightly forward and downward and then (fig. 2 *C*) to the side toward one shoulder, with the nasal tip held slightly upward to cause the liquid contents of the applicator to flow readily out of the open end (*d*) of the nozzle tube into the middle meatus. The handle (*c*) allows leverage to be applied to the applicator, aiding the patient to determine quickly the proper inclination of the head required to deliver the liquid into the middle meatus. The medicinal fluid remains in the infundibulum, which acts like a gutter. There is no overflowing into the pharynx or out of the pharynx or out of the nostril if moderate amounts of fluid are used.

As a final step, the patient's head is brought face downward for the purpose of permitting the medicinal solution to flow out again.

That the solution can be easily delivered into the middle meatus and into the infundilulum by this method was evidenced by a series of roentgenograms, taken in each instance before and after the instillation of iodized poppyseed oil 28 per cent into the nasal cavity by means of the new tilting applicator. These roentgen films were made with the patient lying in a lateral position on the table, with the nostril to be treated downward. In most cases there was no premedication with vasoconstrictors or any other drugs.

One patient received a premedication of 5 drops of 0.25 per cent phenylephrine hydrochloride N.N.R. (neo-synephrine hydrochloride®) delivered with the tilt applicator into the right nostril in order to shrink down the mucous membrane of the middle meatus.

The roentgenograms are shown in figure 3. Figure *A* and *C* were taken ten minutes after the administration of the vasoconstrictor and before the instillation of 0.5 cc. of iodized poppyseed oil 28 per cent. *D*, was taken five minutes before *B* was made.

For more than five minutes the oil seems to have remained in the area of the middle meatus. The inferior meatus does not show any trace of the oil. The same result was achieved by the administration of 1 cc. of iodized poppyseed oil 28 per cent to the right nostril in a number of cases.

SUMMARY

Evidence is presented to demonstrate that an oily or aqueous medicinal solution may be introduced into the middle meatus and into the infundibulum by means of a specially designed nasal applicator. The administration of the solution can be carried out by the patient or by the physician simply, quickly and comfortably, as it requires only the instillation of the tilt applicator and a lateral inclination of the head.

3 East Sixty-Sixth Street.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION, THE AMERICAN MEDICAL ASSOCIATION

As of June 1, 1949

Acousticon Model A-100 — J. A. M. A. **138:** 293 (Sept. 25) 1948.
Mfr. Dictograph Products, Inc.
580 Fifth Avenue
New York 19

Aurex (Semi-Portable) — J. A. M. A. **109:** 585 (Aug. 21) 1937.

Aurex Model C-B and Model C-A — J. A. M. A. **120:** 535 (Oct. 17) 1942.

Aurex Model F — J. A. M. A. **138:** 294 (Sept. 25) 1948.

Aurex Model H — J. A. M. A. **136:** 1099 (April 24) 1948.
Mfr. Aurex Corporation
1117 North Franklin Street
Chicago

Beltone Mono-Pac — J. A. M. A. **130:** 637 (March 9) 1946.

Beltone Harmony Mono-Pac — J. A. M. A. **133:** 543 (Feb. 22) 1947.
Mfr. Beltone Hearing Aid Company
1450 West Nineteenth Street
Chicago

Dysonic Model 1 — J. A. M. A. **137:** 1534 (Aug. 21) 1948.
Mfr. Dynamic Hearing Aids, Inc.
1042 Atlantic Avenue
Brooklyn 16

Electroear Model C — J. A. M. A. **136:** 769 (March 13) 1948.
Mfr. American Earphone Company, Inc.
10 East Forty-Third Street
New York 17

Gem Hearing Aid Model V-35 — J. A. M. A. **139:** 229 (Jan. 22) 1949.
Mfr. Gem Ear Phone Company, Inc.
50 West Twenty-Ninth Street
New York 1

Maico Type K — J. A. M. A. **129:** 32 (Sept. 1) 1945.

Maico Atomeer Mfr. The Maico Company, Inc. North Third Street Minneapolis 1	J. A. M. A. **133:** 542 (Feb. 22) 1947.
Mears Aurophone Model 200	J. A. M. A. **138:** 428 (Oct. 9) 1948.
1947-Mears Aurophone Model 98 Mfr. Mears Radio Hearing Device Corporation 1 West Thirty-Fourth Street New York 1	J. A. M. A. **137:** 1535 (Aug. 21) 1948.
Micronic Model 101 (Magnetic Receiver) Mfr. Micronic Company 727 Atlantic Avenue Boston 11	J. A. M. A. **139:** 99 (Jan. 8) 1949.
Microtone T-3 Audiomatic	J. A. M. A. **138:** 1229 (Dec. 25) 1948.
Microtone T-4 Audiomatic	J. A. M. A. **136:** 109 (Jan. 10) 1948.
Microtone T-5 Audiomatic Mfr. The Microtone Company Ford Parkway on Mississippi St. Paul 5	J. A. M. A. **139:** 379 (Feb. 5) 1949.
National Cub Model	J. A. M. A. **138:** 295 (Sept. 25) 1948.
National Standard Model	J. A. M. A. **138:** 295 (Sept. 25) 1948.
National Star Model Mfr. National Hearing Aid Laboratories 815 South Hill Street Los Angeles 14	J. A. M. A. **138:** 295 (Sept. 25) 1948.
Otarion, Model A-1	J. A. M. A. **115:** 1101 (Sept. 28) 1940.
Otarion, Model A-3	J. A. M. A. **132:** 1071 (Dec. 28) 1946.
Otarion, Models A-4 J & S	J. A. M. A. **132:** 925 (Dec. 14) 1946.
Otarion, Model E-1	J. A. M. A. **136:** 108 (Jan. 10) 1948.
Otarion, Model E-1S	J. A. M. A. **138:** 650 (Oct. 30) 1948.
Otarion, Model E-2	J. A. M. A. **138:** 887 (Nov. 20) 1948.
Otarion, Model E-4 Mfr. Otarion Hearing Aids 159 North Dearborn Street Chicago	J. A. M. A. **140:** 156 (May 14) 1949.
Paravox Model XT	J. A. M. A. **132:** 79 (Sept. 14) 1946.
Paravox Model XTS	J. A. M. A. **134:** 365 (May 24) 1947.
Paravox Models VH and VL	J. A. M. A. **136:** 109 (Jan. 10) 1948

Paravox Model Y (YM, YC and YC-7) Mfr. Paravox, Inc. 2056 East Fourth Street Cleveland 15	J. A. M. A. **140:** 155 (May 14) 1949.
Precision Table Hearing Aid Mfr. Precision Electronics Company 850 West Oakdale Avenue Chicago 14	J. A. M. A. **139:** 785-6 (Mar. 19) 1949.
Radioear Model 45-CM	J. A. M. A. **126:** 1151 (Dec. 30) 1944.
Radioear Model 45-M-magnetic air conduction receiver	J. A. M. A. **127:** 219 (Jan. 27) 1945.
Radioear Model 45-M-magnetic bone conduction receiver	J. A. M. A. **127:** 27 (Jan. 6) 1945.
Radioear Permo-Magnetic Uniphone	J. A. M .A. **139:** 787 (March 19) 1949.
Radioear Permo-Magnetic Multipower Mfr. E. A. Myers & Sons 306 Beverly Road Mount Lebanon Pittsburgh	Report not yet published.
Ravox (Semi-Portable) Mfr. Zenith Radio Corporation 6001 West Dickens Avenue Chicago	J. A. M. A. **113:** 18 (Oct. 28) 1939.
Silver Micronic Hearing Aid, Model 101	J. A. M. A. **135:** 159 (Sept. 20) 1947.
Silver Micronic Hearing Aids Models 202M & 202C Mfr. Micronic Company 727 Atlantic Avenue Boston 11	J. A. M. A. **139:** 848 (March 26) 1949.
Sonotone Audicles Nos. 530 531 and 533	J. A. M. A. **123:** 837 (Nov. 27) 1943.
Sonotone Model 600	J. A. M. A. **131:** 523 (June 8) 1946.
Sonotone Model 700	J. A. M.A. **135:**838 (Nov. 29) 1947.
Sonotone Model 900 Mfr. Sonotone Corporation Elmsford, N. Y.	J. A. M. A. **139:** 786 (March 19) 1949.

Superfonic Hearing Aid Mfr. American Sound Products Inc. 2454 South Michigan Avenue Chicago	J. A. M. A. **138:** 293 (Sept. 25) 1948.
Televox Model E Mfr. Televox Mfg. Company 117 South Broad Street Philadelphia 7	Report not yet published.
Telex Model 22	J. A. M. A. **134:** 605 (June 14) 1947.
Telex Model 97	J. A. M. A. **138:** 294 (Sept. 25) 1948.
Telex Model 612	J. A. M. A. **114:** 1634 (April 27) 1940.
Telex Model 900	J. A. M. A. **117:** 1978 (Dec. 6) 1941.
Telex Model 1020	J. A. M. A. **117:** 2072 (Dec. 13) 1941.
Telex Model 1550 Mfr. Telex, Inc. Minneapolis 1	J. A. M. A. **126:** 705 (Nov. 11) 1944.
Tonemaster Model Royal Mfr. Tonemasters, Inc. 1627 Pacific Avenue Dallas 1, Texas	J. A. M. A. **139:** 848 (March 26) 1949.
Trimm Vacuum Tube Model 300 Mfr. Trimm, Inc. 400 West Lake Street Libertyville, Ill.	J. A. M. A. **133:** 542 (Feb. 22) 1947.
Unex Model A Mfr. Nichols & Clark Hathorne, Mass.	J. A. M. A. **134:** 254 (May 17) 1947.
Vacolite Model J Mfr. Vacolite Company 3003 North Henderson Street Dallas 6, Texas	Report not yet published.
Western Electric Ortho-tronic Model	J. A. M. A. **121:** 1283 (April 17) 1943.
Western Electric Model 63	J. A. M. A. **131:** 895 (July 13) 1946.
Western Electric Model 64	J. A. M. A. **134:** 605 (June 14) 1947.
Western Electric Models 65 & 66 Mfr. Western Electric Company Inc. 120 Broadway New York 5	J. A. M. A. **137:** 534 (June 5) 1948.

Zenith Radionic Model A-2-A J. A. M. A. **127:** 159 (Jan. 20) 1945.
Zenith Radionic Model A-3-A J. A. M. A. **127:** 159 (Jan. 20) 1945.
Zenith Radionic Model B-3-A J. A. M. A. **127:** 158 (Jan. 20) 1945.
Zenith **Model 75** J. A. M. A. **135:** 773 (Nov. 22) 1947.

Mfr. Zenith Radio Corporation
6001 Dickens Avenue
Chicago

All the accepted hearing devices employ vacuum tubes.

Abstracts from Current Literature

Ear

THE CIRCULATION OF THE LABYRINTHINE FLUIDS: EXPERIMENTAL INVESTIGATION IN RABBITS. FRANZ ALTMANN and JULES G. WALTNER, Ann. Otol., Rhin. & Laryng. **56**:684 (Sept.) 1947.

The authors review the work which has been done on the physiology of the labyrinthine fluid. It is generally accepted that the greater part of the endolymph is formed in the stria vascularis of the cochlear duct. Other sources probably exist, although nothing definite is known about them. Resorption of the endolymph is thought by some to take place mainly in the endolymphatic sac, but destruction of this structure and the medial portion of the endolymphatic duct produced no histologic changes in the membranous labyrinth; so it is impossible that the endolymphic sac is the only point of resorption.

The origin and ultimate fate of the perilymph is not clear, either. Some think it is only cerebrospinal fluid which has entered the perilymphatic spaces via the cochlear aqueduct; others think it is derived from endolymph which has diffused through Reissner's membrane; still others think it originates in the blood vessels of the cochlea. Some maintain that it is derived partly from cerebrospinal fluid and partly from certain structures within the inner ear—either from the endolymph or from the blood within the modiolus.

In an attempt to clarify some of these points, the authors injected 0.5 to 1.0 cc. of 1 to 1.66 per cent potassium ferrocyanide and ferric ammonium citrate into the cisterna cerebellomedullaris of rabbits, after withdrawing the same amount of cerebrospinal fluid. They describe their results and, with certain reservations, come to the following conclusions:

The perilymphatic fluid in rabbits is derived mainly from cerebrospinal fluid which reaches the perilymphatic spaces chiefly through the aqueductus cochleae and to some extent through the internal auditory meatus. In view of the fact that Reissner's membrane is so easily penetrated by solutions of iron salts, the possibility exists that some of the perilymph is derived from the endolymph. In the experimental animals, however, the iron salts penetrated from the perilymphatic into the endolymphatic space of the cochlea. If the pathway followed by the iron salts is indicative of the physiologic currents within the perilymph, it can be stated that the resorption of the perilymph within the cochlea takes place at a very rapid rate in the ligamentum spirale and the crista spiralis. No conclusions can be drawn concerning the fate of the perilymph in other parts of the labyrinth. The same evidence makes it probable that resorption of endolymph also occurs at the same places within the cochlea. This would explain the lack of dilatation of the endolymphatic system after obliteration of the endolymphatic sac. In view of the great difference in the structure between the aqueductus cochleae and the modiolus in rabbits and those in man, conclusions based on these experiments in rabbits are not necessarily valid for man.

M. V. MILLER, Philadelphia.

THE REPORT OF A CASE OF CLOSURE OF A POSTAURICULAR MASTOID FISTULA. MAURICE M. PIERCE, Laryngoscope **57**:501 (July) 1947.

Pierce describes a method of closure of a postauricular mastoid fistula which is easily performed and which gives a good cosmetic result. The mastoid cavity is revised and then filled with bone chips taken from the crest of the ilium. The skin, which has been undermined, is closed. Healing takes place without leaving a depression.

HITSCHLER, Philadelphia.

THE PRACTICAL ANATOMICAL AND SURGICAL CONSIDERATIONS FOR EXPOSURE OF THE FACIAL NERVE. FRANK D. LATHROP, Laryngoscope **58**:743 (Aug.) 1948.

Lathrop gives a detailed description of the surgical anatomy of the facial nerve and tells how to find it and how to avoid injuring it. He explains the methods of exposure of the facial nerve in the face, neck and temporal bone.

HITSCHLER, Philadelphia.

COMMENTS ON THE RESULTS OF TREATMENT WITH THE NASAL RADIUM APPLICATOR. A. H. HILDING, Laryngoscope **58**:1103 (Oct.) 1948.

Hilding presents a good review of nasopharyngeal irradiation, with results in a series of 50 cases. The cases are divided into groups according to the diagnosis, and the results obtained in each group noted; these results are particularly interesting.

HITSCHLER, Philadelphia.

Pharynx

HEMANGIOMAS OF THE MOUTH. FREDERICK A. FIGI, Ann. Otol., Rhin. & Laryng. **56**:853 (Dec.) 1947.

Hemangiomas are often encountered about the face and neck and may extend into the oral cavity, but hemangiomas limited to the interior of the mouth are rare. These tumors are congenital or appear shortly after birth and in general are divided into capillary and cavernous types, both of which are subdivided. Capillary hemangiomas include the port-wine stain, or nevus flammeus, and the strawberry birthmark of the hemangioma simplex; the former may involve the oral mucosa, but the latter has not been seen by the author. Cavernous hemangiomas often occur in adults as small, varix-like masses at the vermilion border of the lips or in the oral mucosa and may be due to trauma. The congenital forms may be large and cause enlargement and distortion of the involved parts. Closely allied to the cavernous type are the plexiform or racemose hemangiomas, but these are less common and are probably cavernous hemangiomas with single or multiple arteriovenous fistulas. They are expansile, pulsating tumors, which may be diffuse and of considerable extent. They can be only partially obliterated by pressure and become tenser than simple cavernous angiomas when the patient strains or the part is dependent. A bruit is usually audible and a thrill frequently palpable. Because of arterial pressure they are firmer than the simple cavernous hemangiomas and tend to progress. Phleboliths are commonly found in cavernous hemangiomas in adults and vary from rather soft consistency to calcareous hardness and range in diameter from 2 to 3 mm. to more than 1 cm.

Since the vessels composing the strawberry type of capillary hemangioma are lined with the infantile form of endothelium, they respond well to irradiation. The author prefers this method to excision or the use of solid carbon dioxide and says that it produces a minimum of scarring. Port-wine stains are largely lined with a flattened, adult type of cell, and the cells give little or no response to irradiation.

If radiation is used, many of these later show malignant degeneration. Cosmetics are best used to hide the stain. Sometimes excision with skin grafting is used. A nevus flammeus of the lip associated with a cavernous hemangioma which is causing enlargement and deformity should be controlled by electrocoagulation and by the injection of a sclerosing solution before excision and skin grafting.

In general, irradiation is the treatment of choice for strawberry birthmarks and cavernous hemangiomas in infants and small children. In adults, cavernous lesions are more effectively treated with electrocoagulation and injections of boiling water or sclerosing agents. Well circumscribed hemangiomas often are best excised. Plexiform or racemose hemangiomas usually require ligation of the afferent vessels, in addition to the measures employed in treating cavernous hemangiomas.

MILLER, Philadelphia.

EDEMA OF THE UVULA FOLLOWING DEMEROL-HYOSCINE ANALGESIA. V. R. MOORMAN and I. M. BIRENBOIM, Laryngoscope **59**:66 (Jan.) 1949.

Edema of the uvula following scopolamine medication is rare but has been reported (9 cases, including this one). It is a problem primarily encountered on the obstetric service. (Many otolaryngologists use scopolamine for pretonsillectomy analgesia.—W. J. H.) The edema includes the glottis and usually is so severe that tracheotomy is necessary for relief.

HITSCHLER, Philadelphia.

POSTOPERATIVE TONSIL BLEEDING: STUDIES OF THE ETIOLOGY, PREVENTION AND MANAGEMENT. H. GRANT PRESTON, Laryngoscope **59**:251 (March) 1949.

Preston comprehensively discusses tonsillar bleeding, its prevention and control. The postoperative use of vitamin K greatly reduces the incidence of hemorrhage. Ascorbic acid may also be useful. On the other hand, the use of large amounts of acetylsalicylic acid or of gum containing this agent or undue operative trauma may increase the likelihood of this complication. Many other factors are discussed.

HITSCHLER, Philadelphia.

Larynx

COMPRESSION OF THE TRACHEA BY AN ANOMALOUS INNOMINATE ARTERY: AN OPERATION FOR ITS RELIEF. ROBERT E. GROSS and EDWARD B. D. NEUHAUSER, Am. J. Dis. Child **75**:570 (April) 1948.

Working in the departments of surgery and radiology at Harvard Medical School, Gross and Neuhauser have reported previously on surgery of the chest in children. Hitherto, anomalies of the aortic arch and associated vessels have been medical curiosities only, but now that the radiologist with contrast mediums can give an accurate diagnosis, some hope of surgical relief can be afforded in certain selected instances. A baby weighing 8 pounds (3,628.5 Gm.) was born June 2, 1946, by forceps delivery. There were no noticeable defects, but regurgitation or vomiting was persistent for four months before an accurate diagnosis was made by roentgenogram. There was noisy wheezing dyspnea, relieved only by hyperextension of the head. Iodized oil injected into the trachea permitted photographing of the outline of pressure exerted on the trachea from left to right by an anomalous artery. The operation was undertaken October 17, with the patient under cyclopropane anesthesia, positive pressure being maintained in the lungs by means of a tight-fitting face mask. After operation, the technical details of which are described, the chest was closed and the lung reexpanded fully with subsequent improvement in respiration. The patient was discharged from the hospital on the eighth day!

[Comment: With obstruction of the respiratory tract from whatever cause, infections are often fatal. Therefore tracheal obstruction, especially, must be relieved if a plan of operative procedure can be laid out. The operation described "was not particularly difficult." Explorations of the infant chest are "major" in importance, and demand "adequate facilities" in every detail.]

VOORHEES, New York.

Nose

PENICILLIN THERAPY IN CHRONIC SUPPURATIVE SINUSITIS. A REPORT OF 22 CASES. HAROLD BOYD, Laryngoscope **57**:460 (July) 1947.

Boyd reviews the literature on the treatment of sinusitis with penicillin. There is little doubt that penicillin aids in the treatment of the acute form and in conjunction with sinus surgery. He presents the results of penicillin therapy in 22 cases of chronic sinusitis, in all of which some previous treatment other than with penicillin had been used. Each patient was thoroughly studied before treatment was begun and checked thirty days after treatment was ended. Roentgenograms and bacteriologic studies, including tests for sensitivity to penicillin, were made. Local treatment, including surgical procedures when necessary, was performed. Twenty-thousand units of pencillin was given hypodermically every fourth hour. The results were good almost without exception.

HITSCHLER, Philadelphia.

VASOMOTOR RHINITIS: ITS PHYSIOPATHOLOGIC MECHANISM; EFFECTS OF THE PROCAINE HYDROCHLORIDE INFILTRATION OF THE TEMPORAL ARTERIES. O GODIN and W. CLOETENS, Acta oto-rhino-laryng. belg. **1**:3, 1947.

The authors classify the patients as of five types, according to the pathogenesis of the nasal syndrome:

Allergic Type.—Here the nasal blockage, sneezing and watering are due solely to extraneous substances—pollens, dusts, etc. No permanent circulatory systemic disturbances are present in this group. The antigen contained in the inhalant is neutralized by antibody, producing histamine, which brings about passive dilatation of capillaries and terminal arterioles and constriction of terminal veins. The effect is that of stasis, edema by transudation and increased peripheral venous pressure. The treatment of this type is: (1) systemic—desensitization and administration of a double salt of calcium bromide-galactogluconate (calcibronat®) and antihistaminic agents (N,N-dimethyl-N′-phenyl-N′-benzylethylenediamine [antergan,® or 2339 R.P.] and N,N-dimethyl-N′-[p-methoxybenzyl]-N′-[α-pyridyl] ethylenediamine [neo-antergan,® or 2786 R. P. "Special"]); (2) local—a therapy combining the antihistamine action of 2-(N-phenyl-N-benzylaminomethyl) imidazoline (antistine®) with the constrictor effect of naphazoline hydrochloride (privine hydrochloride®). No procaine hydrochloride infiltration of the type described in the following section is indicated here.

Circulatory Type.—The nasal symptoms are protracted and are made worse by changes of posture, of external temperature and of barometric pressure. Often no specific cause is found, and the allergy tests are not contributory. This group comprises the largest number of patients. In this group associated systemic circulatory disturbances are common—chilliness, cold and cyanotic conditions of the extremities, frostbite. The nasal mucosa is pale and boggy, frequently showing polypoid degeneration. The authors find that in this group the peripheral arterial pressure is normal but the peripheral venous pressure is markedly low (they use Villaret's technic with Claude's water manometer, connected by a 40 cm. rubber

tube filled with sodium citrate solution to a vein of a forearm). They suggest, as the physiopathologic mechanism in this group, a spastic constriction of terminal arterioles with resulting dilatation of capillaries and terminal venules. The histamine does not produce this capillary effect, as it does in patients with hay fever of pure allergic type. This circulatory mechanism is comparable to Raynaud's disease (second stage). The treatment comprises: (1) administration of 1-(p-hydroxyphenyl)-2-methylaminoethanol tartrate (sympatol®), a peripheral vasodilator, which, used alone, gave the authors 9 cures, 4 improvements and 3 failures in 16 patients; (2) crossed endocrine therapy (estrogens in males and testosterone in females), which enhances the effect of the aforementioned drug; (3) procaine hydrochloride infiltration of the sympathetic nerve plexus of the temporal arteries. It is performed by injecting 4 to 5 cc. of a 1 per cent procaine hydrochloride solution without epinephrine hydrochloride into the subcutaneous tissue surrounding the temporal artery on both sides. The immediate effect is marked shrinking of the turbinal mucosa, lasting from six hours to several days; repeated injections bring a lasting effect of several months. At the same time, this procainization of the sympathetic nerve plexus suppresses the generalized arteriolar spasm and brings a return of normal systemic venous pressure.

Endocrine Type.—Menstrual disturbances are associated (amenorrhea, dysmenorrhea); allergy tests are not contributory; peripheral venous pressure is below normal. The nasal symptoms, mostly intermittent, coincide often with the onset of an irregular menstrual cycle or with the onset of the menopause. The pathogenesis appears to be in variations of the blood level of estrogen, causing arteriolar spasm and fall of pressure in terminal veins. This type requires estrogenic treatment and benefits also from procaine hydrochloride infiltration of the sympathetic nerve plexus of the temporal arteries.

Mixed Endocrine-Circulatory Type.—Here the nasal symptoms are more permanent than in the previous group; they occur mostly after the menopause. The pathogenesis is in peripheral arteriolar constriction, and the systemic signs are those of the two preceding groups combined. The treatment consists in use of sympatol® and estrogens and temporal procaine hydrochloride infiltration.

Constitutional Type.—Allergic nasal symptoms appear early in childhood; other allergic manifestations, particularly bronchial asthma, are often associated. The allergic background is inherited. Endocrine and circulatory disturbances, such as cyanosis and coldness of the extremities, are common. In this group the peripheral venous pressure was found increased in contrast to the previous types. The pathologic mechanism here is apparently a loss of tonicity of the capillaries. The nasal symptoms are induced as well by the extraneous allergens as by the circulatory factors (arteriolar spasm). The treatment of this type is mostly symptomatic; estrogens, calcibronat® and ephedrine are helpful; procaine hydrochloride infiltration gives inconstant improvements and influences the associated bronchial asthma more than the vasomotor rhinitis.

HERSON, Chicago.

Miscellaneous

DIPHTHERIA OF THE ESOPHAGUS: A CASE INVOLVING THE STOMACH AND TONGUE. ARCHIBALD L. HOYNE, Am. J. Dis. Child. **74**:80 (July) 1947.

A white girl, born on Jan. 18, 1946, was admitted to the Municipal Contagious Disease Hospital, Chicago, on February 20. The diagnosis at examination was pharyngeal and tonsillar diphtheria, with profound toxemia and bronchopneumonia. In spite of all treatment, the patient died five hours after admission. There was

no history of exposure to diphtheria. While cultures from the child's nose and throat yielded diphtheria organisms, cultures obtained from both parents were sterile. Autopsy showed that a continuous, tough, grayish membrane extended from the base of the tongue down the entire length of the esophagus and protruded into the stomach like a flower. The inner surfaces of the larynx, trachea and bronchi were free from exudate.

The author comments on the relative rarity of diphtheria of the esophagus and of the stomach and states that such a diagnosis is not likely to be made during the life of the patient. However, a careful examination of the esophagus in cases of severe pharyngeal diphtheria might result in more frequent reports of this condition. How a 33 day old baby, breast fed exclusively, contracted diphtheria without known exposure or contact was not determined.

VOORHEES, New York.

ONE STAGE PHARYNGEAL DIVERTICULECTOMY. T. A. SHALLOW and L. H. CLERF, Surg., Gynec. & Obst. **86**:317 (March) 1948.

Preference is expressed for a one stage surgical procedure with the simultaneous teamwork of surgeon and esophagoscopist. Preoperative preparation is directed toward correction of nutritional deficiencies and detection of concomitant disease. In the operative approach, the esophagoscope is employed for emptying the sac and for the maintenance of the normal anatomic position of the pharynx and upper part of the esophagus throughout the operation. Open drop ether anesthesia is used.

Introduction of the esophagoscope into the subdiverticular portion of the esophagus is facilitated by the use of an olive-tipped bougie passed over a previously swallowed string. At the conclusion of the operation, a feeding tube is inserted through the esophagoscope into the lower portion of the esophagus. After amputation of the sac, the stump is transplanted upward, beneath the inferior constrictor muscle. The muscular defect in the pharyngeal wall is corrected by approximating the upper margin of the cricopharyngeus to the lower margin of the inferior constrictor muscle. This suture line is anchored posteriorly to the prevertebral fascia, thus obliterating the retropharyngeal space and erecting a barrier against descending infection.

In the entire series of 186 cases, there were 5 deaths, a mortality rate of approximately 3 per cent. The authors thus conclude that the mortality for the one stage pharyngeal diverticulectomy is no greater than would be expected for any other major operation in patients of this age group.

FRIEDBERG, Chicago.

Book Reviews

Oral Surgery. By Kurt H. Thoma, D.D.M., Professor of Oral Surgery and Brackett Professor of Oral Pathology, Harvard University; Oral Surgeon and Chief of Dental Department, Massachusetts General Hospital. Two volumes. Price, $30. Pp. 1,521, with 1,631 illustrations, 121 in color. St. Louis: The C. V. Mosby Company, 1948.

It will doubtless come as a surprise to many readers to find a work of over 1,500 pages dealing solely with the mouth, jaws and adjacent structures. (Most physicians could tell in a few words all they know about this subject unless they have read widely or have taken it up as a side line.) But here is a further surprise: The work is notable for its omissions, listed by the author. There is nothing here concerning harelip or cleft palate. These are treated in "Oral Pathology," a previous book written by Dr. Thoma. There is nothing about orthodontia, and "another purposeful omission is the topic of surgical prosthesis." What, then, is offered in this massive text? One can do little more than describe the general layout of the volumes.

The book is based on the assumption that the physician should know all that he can about the patient from a general medical standpoint. To quote Dr. Thoma: "To some, it may seem that supportive care has been overemphasized. I feel, however, that the general treatment is inseparable from the treatment of regional disease . . . the correction of deficiency diseases, the improvement of the general health by diet and rest, general medication to relieve pain, and the administration of drugs to prevent or eliminate infection and decrease the risk of operation, as well as postoperative physiotherapy to improve function." This is, indeed, a large order, but it is sound in plan, even if it may become difficult in practice. Overcoming difficulties is basic in any form of therapeutic procedure. This book was written for the undergraduate in medicine and dentistry and for the graduate student who is interested in dental and oral surgery.

As is fitting, chapter I deals with the principles of surgery, which, of course, are elementary in any such text. Surgical bacteriology, nutritional factors, asepsis and antisepsis, anesthesia and biologic research are discussed. Tracheotomy, procedures of plastic surgery and bone grafting are also described here, but these subjects might be classified to better advantage elsewhere in the book.

Dentoalveolar surgery includes replantation and transplantation of teeth, as well as their extraction. Exodontia, or tooth extraction, has long been a highly specialized field, but it had its origin at any rate in the hands of the old-time general practitioner of medicine.

Chapter III is concerned with treatment of traumatic diseases of the jaws and of fractures of the jaws, but with special reference to infections, such as osteomyelitis. An important phase is the management of delayed union and malunion. In general, fractures of the mandible belong in the field of dentistry, rather than in that of general surgery, since preservation of teeth or loss of them is strictly a dentist's responsibility.

Chapter V will interest the ear, nose and throat specialist, since it deals chiefly with the maxillary sinus. It has long been a matter for serious discussion whether maxillary sinusitis is primary, involving a tooth secondarily, or whether the disease is carried from an infected tooth into the antrum; certainly, either is

possible, and the question is chiefly one of academic interest. The matter of management is paramount. The ear, nose and throat specialist should be grateful for an acknowledgment that oroantral fistulas often follow tooth extraction, with enlargement of the socket by the dentist by use of the curet and the electrically driven burr. This operation is one of the worst in all surgery, since an otherwise normal antrum may be infected from the mouth. Thereafter, pus is drained inadequately from the antrum into the mouth, and no amount of irrigation and attempted "sterilization" can overcome this inadequacy of drainage. The opening is usually at the site of the second cuspid or the first molar. Cure is accomplished by way of a radical operation on the antrum, followed by an effort to produce growth of new tissue to the fistulous opening. If and when the antrum is clean, any one of several procedures in plastic surgery may be performed to "close the hole." One may be grateful, too, for the statement that the Caldwell-Luc operation is preferred, "since the Denker operation frequently interferes with the innervation of the teeth." This is reason enough, but one might add that another reason for its preference is that it is not good surgical procedure to destroy a mass of normal bone—the anatomic pillar of the superior maxilla. Moreover, the Denker operation is unnecessary, since the Caldwell-Lac procedure gives such highly satisfactory results.

Volume II opens with a discussion of the mandibular joint. The joint is affected by the same diseases with which other joints are involved, especially with rheumatoid arthritis, arthritis of infectious origin and ankylosis. Certain aural disabilities are said to be due sometimes to lesions of the mandibular joint. Relaxed ligaments give rise to dislocations and to changes in the synovial fluid, of which there may be too much or too little, this being the diagnosis often made by the rhinologist. Operation on this joint is fraught with many pitfalls, which are mentioned by Dr. Thoma at sufficient length. The clinical varieties of infections of the face and neck following tooth extraction are interesting and significant and are adequately discussed in this volume. The presence of stone in the salivary ducts, dermoid cysts, cysts of the jaws, tumors of the jaws and conditions which involve the fifth and seventh nerves are the principal subjects set forth in this second volume. In chapter XII is discussed benign tumors of the mouth and jaw, while chapter XIII has to do with malignant tumors of these areas. The final chapter (XIV) is concerned with surgical treatment of deformities of the jaw which affect facial symmetry. Both operative and postoperative care are described clearly and completely.

It is probable that this work of Dr. Thoma will be standard for many years to come. So far as is known, there is no other book quite like it in the English language.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Fourth International Congress of Otolaryngology

President: Dr. V. E. Negus, London, England.
General Secretaries: Dr. F. C. W. Capps and Dr. W. A. Mill, 45 Lincoln's Inn Fields, London, W. C. 2.
Place: London. Time: July 18-23, 1949.

Second Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Montevideo. Time: January 1950.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. William H. Johnston, 1515 State St., Santa Barbara, Calif.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit 26, Mich.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles 14.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-26, 1950.

American Laryngological Association

President: Dr. Frederick T. Hill, 177 Main St., Waterville, Maine.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Hotel Mark Hopkins, San Francisco. Time: May 23-24, 1950.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

SECTIONS:

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Southern.—Chairman: Dr. William D. Stimson, 899 Madison Ave., Memphis, Tenn.
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Western.—Chairman: Dr. J. B. Naftzger, 6777 Hollywood Blvd., Hollywood, Calif.

American Otological Society

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Secretary: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 3, N. Y.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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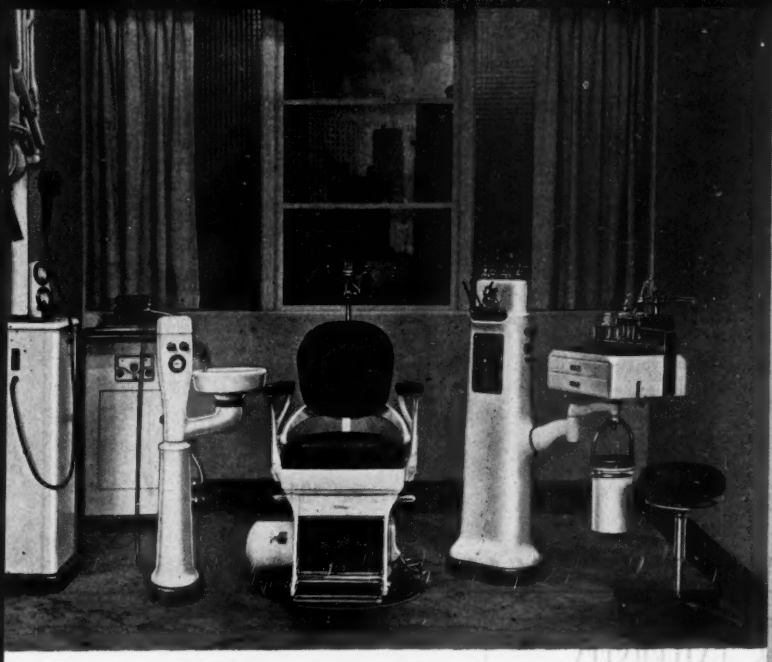

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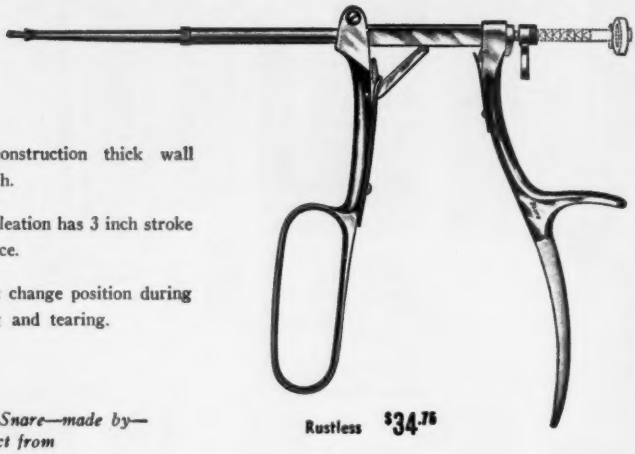

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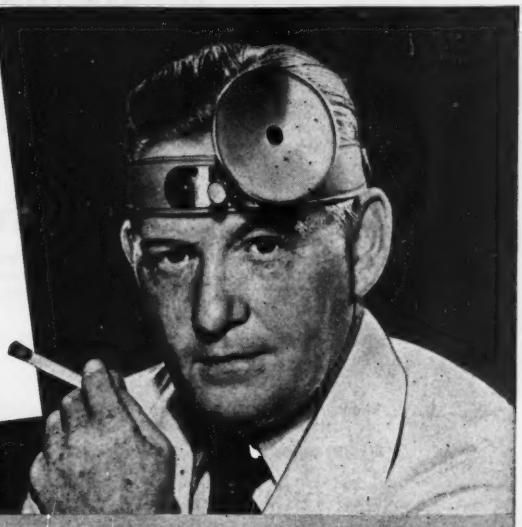

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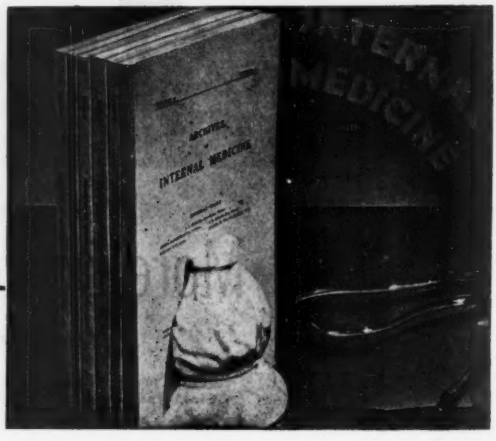

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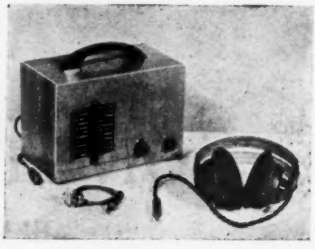

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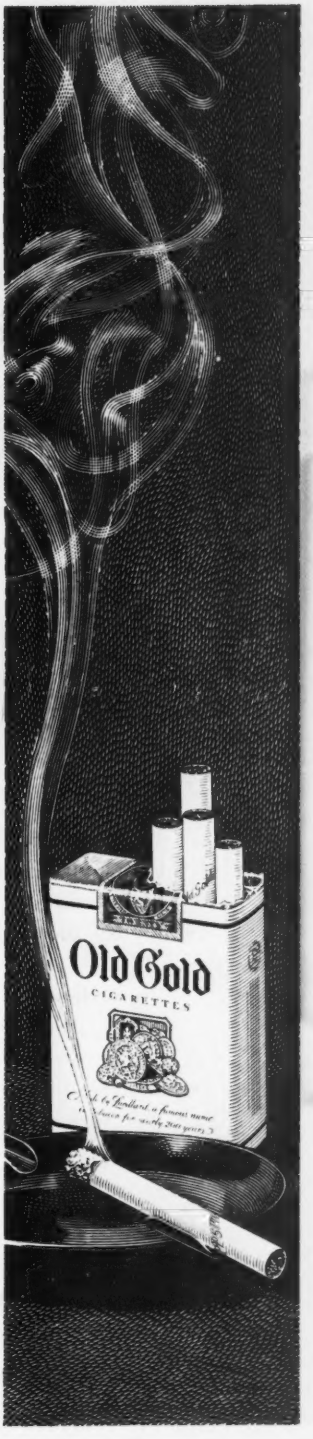

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